HELLP SYNDROME

MATERNAL AND PERINATAL OUTCOME

Dissertation submitted in partial fulfilment of the

Requirement for the award of the Degree of

M.S. DEGREE – BRANCH VI

OBSTETRICS AND GYNAECOLOGY

APRIL 2016

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY,

CHENNAI,

TAMIL NADU

CERTIFICATE

This is to certify that the Dissertation entitled "**HELLP SYNDROME: MATERNAL AND PERINATAL OUTCOME**" submitted by Dr.Krishnaveni, MBBS., DGO., to The Tamilnadu Dr.M.G.R. Medical University, Chennai, in partial fulfilment for the award of M.S (Obstetrics and Gynaecology) is a bonafide work carried out by her under my guidance and supervision during the academic year 2014-2016. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

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DECLARATION

I, Dr.Krishnaveni, MBBS.,DGO., solemnly declare that the Dissertation titled **"HELLP SYNDROME: MATERNAL AND PERINATAL OUTCOME"** had been prepared by me under the expert guidance and supervision of **Prof.Dr.M.Sujatha,MD.,(OG)** Professor, Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital, Tirunelveli.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the regulation for the award of M.S. Degree (Branch VI) in Obstetrics and Gynaecology.

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

Place: Tirunelveli.
Date:

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POST GRADUATE,
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ACKNOWLEDGEMENT

I am very much thankful to the Dean **Dr. K.Sithy Athiya Munavarah**, Triunelveli Medical College Hospital, Tirunelveli, who has granted permission to do this study in this institution,

I take this opportunity to express my deepest sense of gratitude to professor **Dr.MEENA, M.D., DGO., DNB.,** Head of the Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital, Tirunelveli for encouraging me and rendering timely suggestions and guiding me throughout the course of this study. I will be forever indebted to her for her constant support.

I sincerely thank my professor **Dr.RAMALAKSHMI, M.D.,(OG)., Dr.SHEBA ROSATTE VICTOR,M.D.,(OG)., Dr.M.Sujatha,M.D., (OG), Dr.Vijayalakshmi,MD.,DGO.,** for their support and guidance.

I am very much thankful to professor **Dr.M.Saradha,M.D., (Bio-Chemistry)**, Head of Department of Bio-Chemistry for providing valuable support and guiding through the study.

I am extremely thankful to my guide **Dr.M.Sujatha, M.D., (OG)** for guiding me throughout the study.

I am extremely thankful to my registrar **Dr.Tamil Kothai,M.D., (OG)** for her guidance and support throughout my study period in this institution.

I am extremely thankful to all my **Assistant Professors** of the **Department of Obstetrics and Gynaecology** for their guidance and support throughout my study period in this institution.

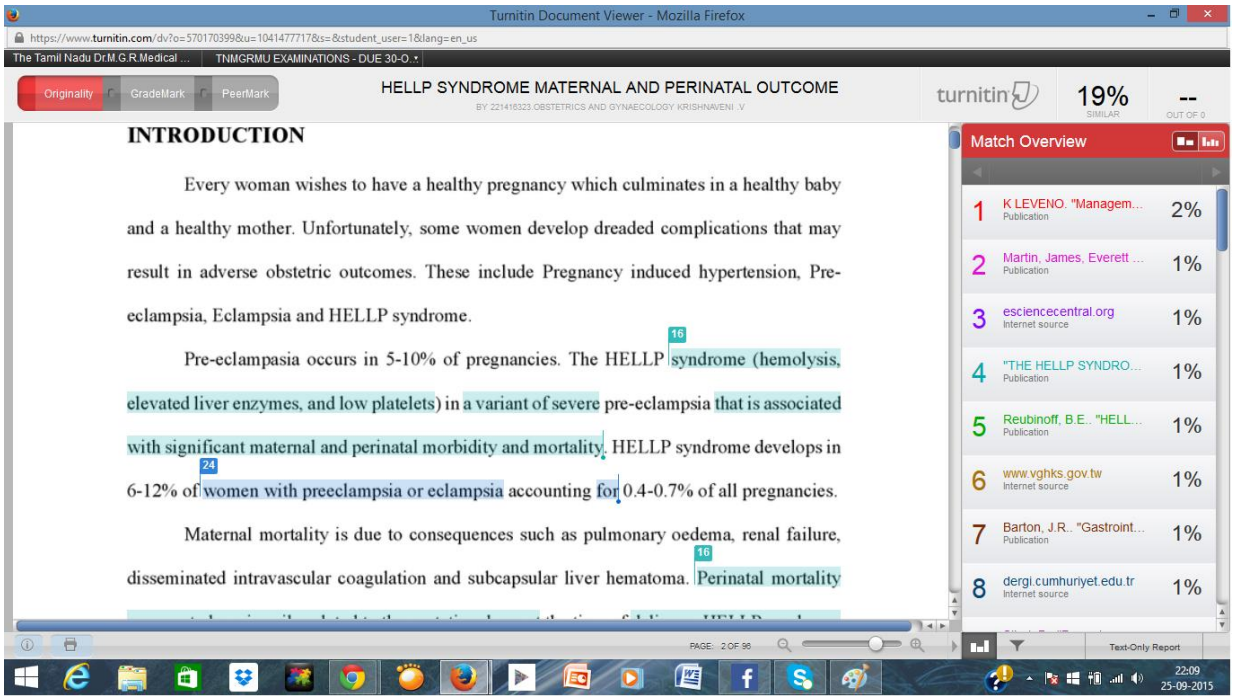
I thank **Prof. P. Arumugam** statistician for their useful inputs.

I wish to express my gratitude to my parents, my brother, my husband and my son & daughter for their support throughout my study.

I also like to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement.

Last but not least, I am very much thankful to all antenatal mothers of Tiruneveli Medical College without whom this study would not have been possible.

CONTENTS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INTRODUCTION
Every woman wishes to have a healthy pregnancy which culminates in a healthy baby and a healthy mother. Unfortunately, some women develop dreaded complications that may result in adverse obstetric outcomes. These include Pregnancy induced hypertension, Pre-eclampsia, Eclampsia and HELLP syndrome.
Pre-eclampsia occurs in 5-10% of pregnancies. The HELLP syndrome (hemolysis, elevated liver enzymes, and low platelets) in a variant of severe pre-eclampsia that is associated with significant maternal and perinatal morbidity and mortality. HELLP syndrome develops in 6-12% of women with preeclampsia or eclampsia accounting for 0.4-0.7% of all pregnancies.
Maternal mortality is due to consequences such as pulmonary oedema, renal failure, disseminated intravascular coagulation and subcapsular liver hematoma. Perinatal mortality
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CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 659/PATHO/2015

PROTOCOL TITLE: HELLP SYNDROME – MATERNAL AND PERINATAL OUTCOME.

PRINCIPAL INVESTIGATOR: DR.V.KRISHNAVENI, MBBS.,

DESIGNATION OF PRINCIPAL INVESTIGATOR: POST GRADUATE IN OBSTETRICS & GYNAECOLOGY.
DEPARTMENT & INSTITUTION: TIRUNELVELI MEDICAL COLLEGE , TIRUNELVELI

Dear ,Dr.V.Krishnaveni , MBBS, The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 09.02.15.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

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Member Secretary, TIREC
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ABSTRACT

BACKGROUND:

A comparative study of fetomaternal outcome in a patients with severe pre-eclampsia and eclampsia with HELLP syndrome and without HELLP Syndrome.

Purpose: To determine the trend of occurrence of HELLP syndrome, risk factors, its complications and its effect on maternal and perinatal outcome of HELLP syndrome in pregnant women at Tirunelveli Medical College Hospital, Tirunelveli.

METHODS:

Patients with severe hypertension in pregnancy who were admitted between Feb2015 to July 2015 at Tirunelveli Medical College Hospital, Tirunelveli.. Out of 100 cases if severe preeclampsia/eclampsia, there were 43 cases of HELLP syndrome. These were compared with case without HELLP syndrome for their mode of presentation along with maternal and perinatal morbidity and mortality.

RESULT:

The overall incidence of HELLP syndrome was 0.4% in the antepartum factors; class V SES, multi (55.8% Vs 33.3%, $p<0.05$), preeclampsia (90% Vs 73.7%, $p<0.05$), symptoms (81.4% vs 61.4%, $p<0.05$), Haemolysis (86% vs 3.5% $p<0.05$), ARF (16.2% vs 3.5% $p<0.05$) were statistically significant. Inthe intrapartum factors there were no significant differences between the two groups in mode of delivery. Abnormal perinatal outcome was (54.5% vs 24.6% $p<0.001$).

Conclusion:

Women with severe hypertension in pregnancy manifesting with HELLP syndrome show a significantly greater frequency of developing Abruption, ARF and DIC. Therefore, their care necessitates intensive monitoring to preclude

development of these complications. Successful management requires recognition, a timely intervention and to render optimal patient treatment.

INTRODUCTION

Every woman wishes to have a healthy pregnancy which culminates in a healthy baby and a healthy mother. Unfortunately, some women develop dreaded complications that may result in adverse obstetric outcomes. These include Pregnancy induced hypertension, Pre-eclampsia, Eclampsia and HELLP syndrome.

Pre-eclampasia occurs in 5-10% of pregnancies. The HELLP syndrome (haemolysis, elevated liver enzymes, and low platelets) in a variant of severe pre-eclampsia that is associated with significant maternal and perinatal morbidity and mortality. HELLP syndrome develops in 6-12% of women with preeclampsia or eclampsia accounting for 0.4-0.7% of all pregnancies.

Maternal mortality is due to consequences such as pulmonary oedema, renal failure, disseminated intravascular coagulation and subcapsular liver hematoma. Perinatal mortality appears to be primarily related to the gestational age at the time of delivery. HELLP syndrome is regarded as a higher risk for the mother and neonate compared to pre-eclampsia.

As our hospital provides treatment facilities to large number of Pre-eclampsia, eclampsia and a relatively higher number of patients of HELLP syndrome, we have the opportunity to conduct such studies which can help us to determine the trend of occurrence of HELLP syndrome, its complications and its effect on maternal and fetal outcome. This will help us in understanding better about the pathophysiology of the disease which can be applied to improve the management and thereby improve the maternal and perinatal outcome.

AIM OF THE STUDY

A comparative study of fetomaternal outcome in a patients with severe pre-eclampsia and eclampsia with HELLP syndrome and without HELLP Syndrome.

To determine the trend of occurrence of HELLP syndrome, risk factors, its complications and its effect on maternal and perinatal outcome of HELLP syndrome in pregnant women at Tirunelveli Medical College Hospital, Tirunelveli.

REVIEW OF LITERATURE

HISTORICAL ASPECTS OF HELLP SYNDROME

In the year 1954 Prichard et al and Chesley (1978) described that pre eclamptic patient are more prone to developed haemolysis, hepatic dysfunction and low platelets[1,3]. But the acronym HELLP Syndrome was coined by Louis Weinstein 1982 (hemolysis, elevated liver enzymes and low platelets) that clinicians could more easily recognise.[6]

Goodlin considered it an early form of severe preeclampsia and labelled as a great imitator, impending gestosis, EPH (edema, proteinuria, hypertension), Gestosis type B and extended toxemia syndrome.[4,5] Weinstein considered it an unique variant of preeclampsia,[6] while Mackenna and Colleagues considered it as misdiagnosed preeclampsia.[7]

CLASSIFICATION OF HYPERTENSIVE DISORDER IN PREGNANCY

According to NHBPEP- National High Blood Pressure Education Program (2000) hypertensive disorder in pregnancy is classified as

1. GESTATIONAL HYPERTENSION:

- New onset hypertension after 20 weeks of pregnancy
- Systolic BP ≥140 or diastolic BP≥90mm Hg for first time during pregnancy
- No proteinuria
- BP returns to normal before 12 weeks postpartum
- Final diagnosis made only postpartum

2. PREECLAMPSIA:

MILD PREECLAMPSIA

- BP≥140/90mm Hg after 20 weeks gestation
- Proteinuria≥300 mg/24 hours or ≥1+dipstick

SEVERE PREECLAMPSIA:

- BP ≥160/110mm Hg
- Proteinuria 2.0g/24 hours or ≥2+ dipstick
- Serum creatinine > 1.2mg/dl unless known to be previous elevated
- Platelets < 100,000/µL
- Microangiopathic hemolysis-increased LDH
- Elevated serum transaminase levels- ALT or AST
- Persistent headache or other cerebral or visual disturbance
- Persistent epigastric pain

3. ECLAMPSIA:

- Seizures that cannot be attributed to other causes in a women with preeclampsia

4. SUPERIMPOSED PREECLAMPSIA ON CHRONIC HYPERETENSION:

- New-onset proteinuria ≥ 300mg/24 hours in hypertensive women but no proteinuria before 20 weeks gestation
- A sudden increase in proteinuria or blood pressure or platelet count < 100,000/µL in women with hypertension and proteinuria before 20 weeks gestation

5. CHRONIC HYPERTENSION:

- BP ≥ 140/90mm Hg before pregnancy or diagnosed before 20 weeks gestation

or

- Hypertension persistent after 12 weeks postpartum

INCIDENCE OF PREECLAMPSIA

- 6-15% in multipara
- 2-4% in multipara[77]

RISK FACTORS FOR PRE-ECLAMPSIA, sibai BM, lancet 2005

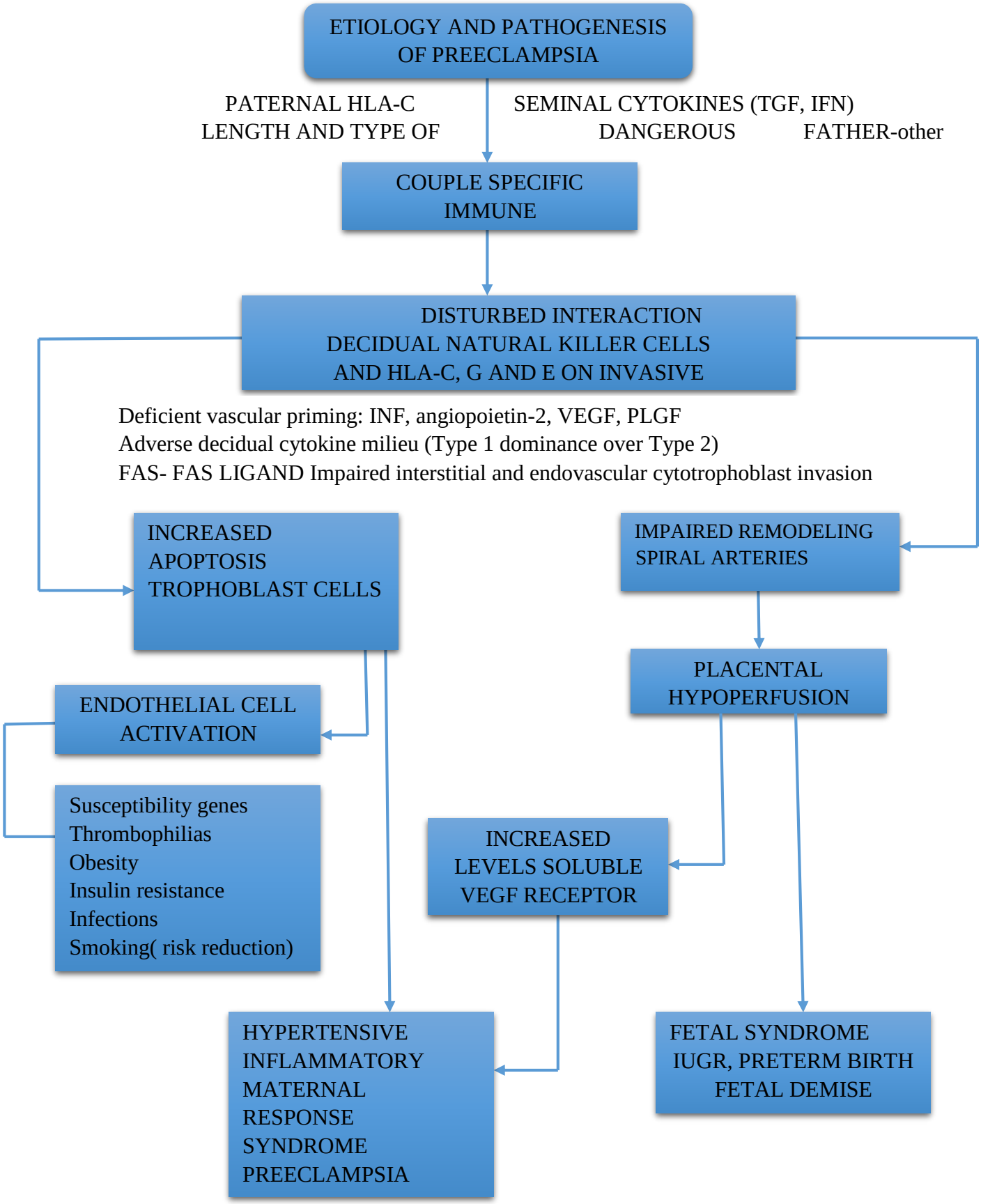
- Age > 35 years
- Parity
- Interval from last pregnancy > 10years
- Obstetric factors
    - preeclampsia or gestational hypertension in prior pregnancy
    - Multiple gestation
    - Hydatidiform mole
    - Hydrops fetails
    - Abnormal uterine artery Doppler at 18-24 weeks
- Family history of pre-eclampsia
- pre-existing medical disorders
    - Hypertension
    - Diabetes melitius
    - Obesity (BMI of 35kg/m^2 or more)

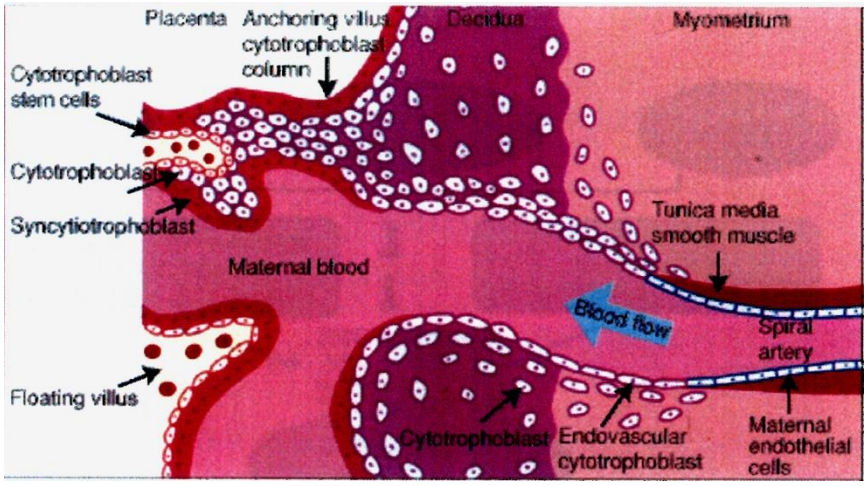
- Renal disease
- vascular disease
- Autoimmune disease
- Thrombophilias

- Couple-related risk factors[34]
  - Primipaternity
  - Limited sperm exposure
  - Pregnancies after donor insemination; oocyte donation; embryo donation
  - Protective effect of "partner change" in case of previous preeclamptic pregnancy
  - "Dangerous male partner" (paternal effects)

ETIO PATHOGENESIS OF PREECLAMPSIA

- ❖ Increased risk of preeclampsia in women exposed to chorionic villi for first time.
- ❖ Hyperplacentosis (abundance of chorionic villi-twins, mole)
- ❖ Genetic predisposition
- ❖ Abnormal trophoblast invasion
- ❖ Immunological maladaptation to inflammatory changes.
- ❖ Calcium and magnesium deficiency[68]


ETIOLOGY AND PATHOGENESIS OF PREECLAMPSIA
PATERNAL HLA-C
LENGTH AND TYPE OF
SEMINAL CYTOKINES (TGF, IFN)
DANGEROUS
FATHER-other
COUPLE SPECIFIC IMMUNE
DISTURBED INTERACTION DECIDUAL NATURAL KILLER CELLS AND HLA-C, G AND E ON INVASIVE
Deficient vascular priming: INF, angiopoietin-2, VEGF, PLGF
Adverse decidual cytokine milieu (Type 1 dominance over Type 2)
FAS- FAS LIGAND Impaired interstitial and endovascular cytotrophoblast invasion
INCREASED APOPTOSIS TROPHOBLAST CELLS
IMPAIRED REMODELING SPIRAL ARTERIES
PLACENTAL HYPOPERFUSION
ENDOTHELIAL CELL ACTIVATION
Susceptibility genes
Thrombophilias
Obesity
Insulin resistance
Infections
Smoking(risk reduction)
INCREASED LEVELS SOLUBLE VEGF RECEPTOR
HYPERTENSIVE INFLAMMATORY MATERNAL RESPONSE SYNDROME PREECLAMPSIA
FETAL SYNDROME IUGR, PRETERM BIRTH FETAL DEMISE


Placenta
Anchoring villus
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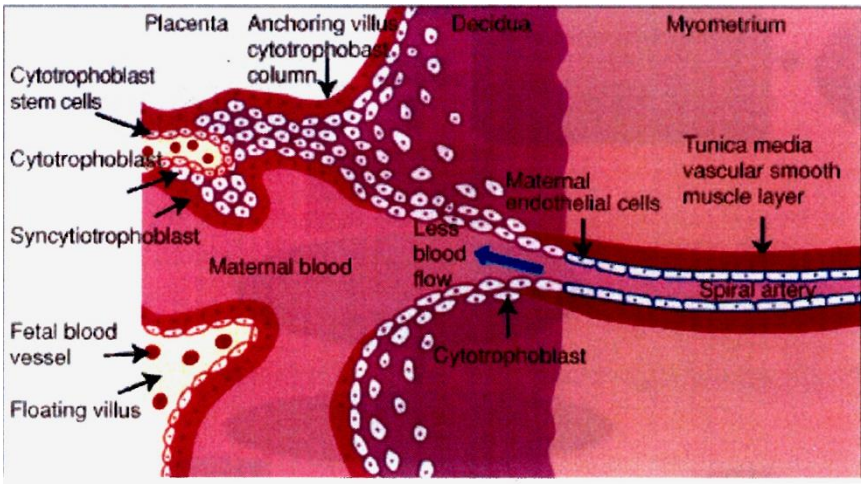
Decidua
Myometrium
Cytotrophoblast stem cells
Cytotrophoblast
Syncytiotrophoblast
Maternal blood
Tunica media smooth muscle
Blood flow
Spiral artery
Floating villus
Cytotrophoblast
Endovascular cytotrophoblast
Maternal endothelial cells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Cytotrophoblast
Syncytiotrophoblast
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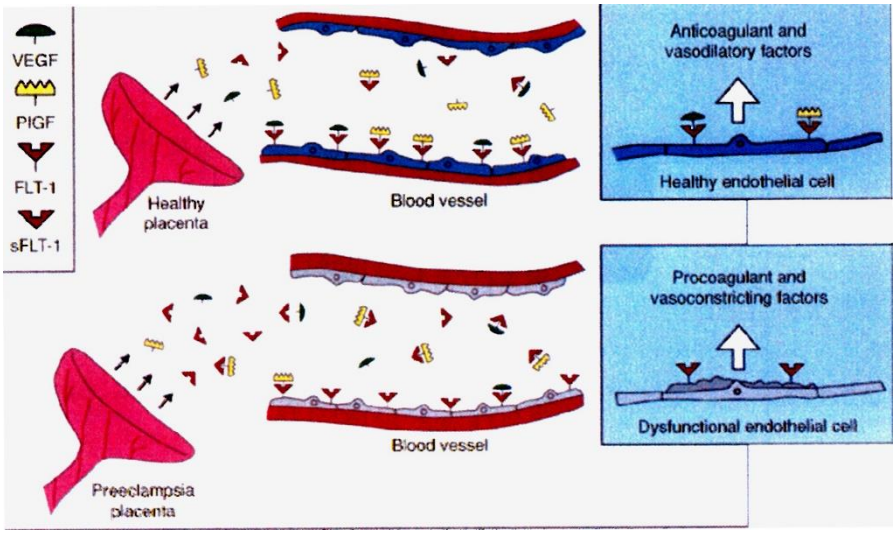
Maternal endothelial cells
Tunica media vascular smooth muscle layer
Less blood flow
Spiral artery
Fetal blood vessel
Floating villus
Cytotrophoblast


VEGF
PIGF
FLT-1
sFLT-1
Healthy placenta
Blood vessel
Anticoagulant and vasodilatory factors
Healthy endothelial cell
Preeclampsia placenta
Blood vessel
Procoagulant and vasoconstricting factors
Dysfunctional endothelial cell

CLINICAL FEATURES

- Severe Head ache
- Vomiting
- Epigastric pain
- Visual disturbances
- Papilledema
- Oliguria
- Thrombocytopenia edema
- HELLP Syndrome

INVESTIGATIONS

- Urine analysis proteinuria.
- Hb-raised(Hemoconcentration, except in hemolysis)
- Platelet-low.
- Peripheral smear-shistiocytes.
- INR and APTT-higher in DIC.
- Serum creatinine – higher
- ALT, AST, LDH-higher
- Albumin-lower
- Bilirubin – higher.
- FUNDUS examination.

PREDICTIVE TESTS FOR DEVELOPMENT OF THE PREECLAMPSIA

Conde – Agudelo & Associates,2009.

PLACENTAL PERFUSION/ VASCULAR RESISTANCE

Roll-over test, isometric handgrip or cold pressor test, angiotensin-II infusion, midtrimester mean arterial pressure, platelet angiotensin-II binding, rennin, 24-hour ambulatory blood pressure monitoring, uterine artery or fetal transcranial Doppler velocimetry

FETEL-PLACENTAL UNIT ENDOCRINE DYSFUNCTION

Human chronic gonadotropin (hCG), alpha-fetoprotein (AFP), estriol, pregnancy associated protein A (PAPP A), inhibin A, activin A, placental protein 13, corticotrophin releasing hormone

RENAL DYSFUNCTION

Serum uric acid, microalbuminuria, urinary calcium or kallikrein, microtransferrinuria, N-acetyl-β-glucosaminidase

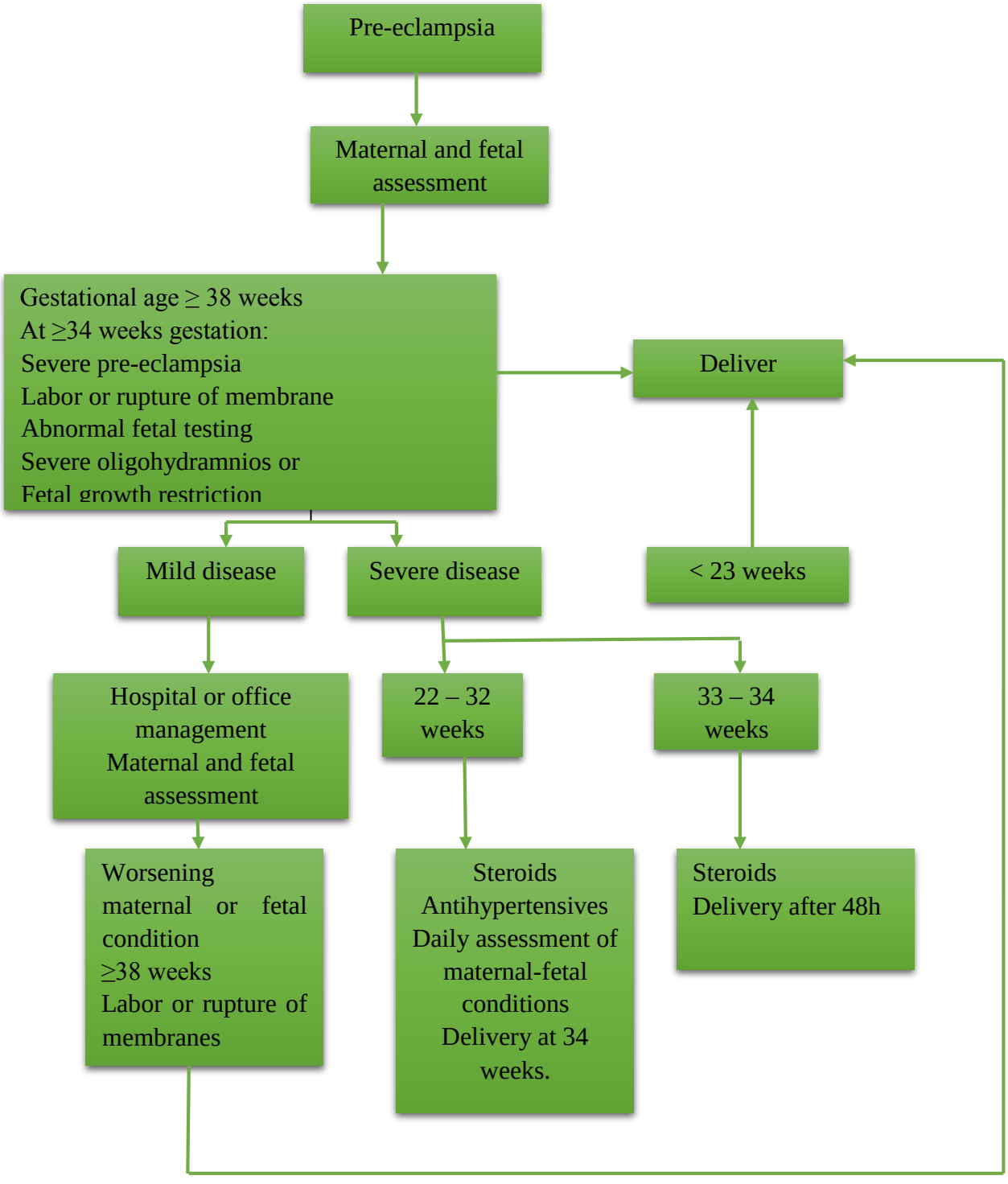
ENDOTHELIAL DYSFUNCTION/ OXIDANT STRESS

Platelet count and activation, fibronectin, endothelial adhesion molecules, prostaglandin, thromboxane, C-reactive protein, cytokines, endothelin, neurokinin B, homocysteine, lipids, antiphospholipid antibodies, plasminogen activator-inhibitor (PAI), leptin, p-selectin, angiogenic factors to include placental growth factor (PIGF), vascular endothelial growth factor (VEGF). fms-like tyrosine kinase receptor-1 (sFIt-1), endoglin[68]

OTHERS/MISCELLANEOUS

Antithrombin-III (AT-3), atrial natriuretic peptide (ANP), β_2 - microglobulin, genetic markers, free fetal DNA, serum proteonomic markers

MANAGEMENT



TIMING FOR DELIVERY (ACOG, 2010)

- 38-39 weeks of gestation for women not requiring medication.
- 37-39 weeks for women with controlled hypertension with medication.
- 36-37 weeks with severe hypertension.
- HYPITAT (Hypertension and Preeclampisa Intervention Trial At Term) (Koopmans et al, 2009) shows the outcome of pregnancy with gestational HT induced at >37 weeks was better compared to expectant management[17].

INDICATIONS FOR DELIVERY WITH EARLY-ONSET SEVERE PREECLAMPSIA FROM SIBAI AND BARTON (2007).

I. MATERNAL

1. Persistent severe headache or visual changes;
2. Pulmonary edema S_aO_2< 94%
3. Uncontrolled severe hypertension despite treatment
4. Oliguria < 500 ml/24 hr or serum creatinine ≥ 1.5 mg/dL
5. Persistent platelet counts<100,000/µL
6. Suspected abruption.

II. FETAL

1. Severe growth restriction - < 5th percentile for EGA
2. Persistent severe oligohydramnios – AFI < 5cm
3. Biophysical profile ≤ 4 done 6 hr apart
4. Reversed end-diastolic umbilical artery flow
5. Fetal death

CONTROL OF BLOOD PRESSURE

CHOICE OF DRUGS

NICE,2010 recommends Labetalol is the first line of drug[29]. It can be given both orally and intravenously. Due to beta receptor blocking activity, it is better to avoid in asthmatic. Labetalol causes neonatal hypoglycaemia and bradycardia.

OTHER DRUGS IN USE ARE

- Nifedepine
- Hydralazine
- Avoid sublingual nifedepine to reduce BP in patient with volume depletion, since it will cause percipitious fall in BP (NICE, 2010).
- Antidote for Mgso4 toxicity is 10 g of 10% calcium gluconate given IV.
- Labetalol 20mg IV bolus followed by 40 mg if not effective within 10 minutes then, 80mg every 10 mins, max dose 220mg.
- Nifedepine-10mg orally every 30 mins, max of 3 doses.
- Hydralazine 5-10mg IV every 15-20 minutes

SEIZURE PREVENTION

Magnesium sulfate dosage schedule dosage schedule for severe preeclampsia and eclampsia

Continuous intravenous infusion, frederick P.Zuspan,MD.,

1. Give 4- to 6-g loading dose of magnesium sulfate diluted in 100mL of IV fluid administered over 15-20 min[54,68]
2. Begin 2g/hr in 100mL of IV maintenance infusion. Some recommend 1g/hr
3. Monitor for magnesium toxicity:

a. Assess deep tendon reflexes periodically

b. Urinary output.

c. Respiratory rate.

4. Magnesium sulfate is discontinued 24 hr after delivery

Intermittent Intramuscular Injections (Pritchard Regimen)

1. Give 4g of magnesium sulfate ($MgSO_4 \cdot 7H_2O$ USP) as a 20% solution intravenously at a rate not to exceed 1g/min
2. Followed by 10 g of 50% magnesium sulfate solution, one-half (5g) injected deeply in the upper outer quadrant of both buttocks through a 3-inch-long 20-gauage needle. (Addition of 1.0mL of 2% lidocaine minimizes discomfort). If convulsions persist after 15 min, give up to 2g more intravenously as a 20% solution at a rate not to exceed 1g/min.
3. Every 4 hr thereafter give 5g of a 50% solution of magnesium sulfate injected deeply in the upper outer quadrant of alternate buttocks, but only after ensuring that:
    a. The patellar reflex is present
    b. Respirations are not depressed, and
    c. Urine output the previous 4hr exceed 100mL
4. Magnesium sulfate is discontinued 24 hr after delivery

COMPLICATION

MATERNAL COMPLICATIONS OF SEVERE PREECLAMPSIA

RESPIRATORY SYSTEM

- Pulmonary edema 2-5%

CNS

- Eclampsia <1%.
- Hemorrhage.
- Cortical blindness.
- Retinal blindness.
- Raised ICT.
- Encephalopathy.

RENAL SYSTEM

- Cortical necrosis
- Renal tubular necrosis 1-5%

CVS

- Left ventricular failure
- Aortic dissection

LIVER

- HELLP syndrome 10-20%
- Subcapsular hematoma <1%

COAGULATION SYSTEM

- DIC
- Hemorrhage

MULTIPLE ORGANS

- MODS
- Death

HELLP SYNDROME

INCIDENCE OF HELLP SYNDROME

Overall incidence of HELLP Syndrome is one in thousand pregnancies incidence in pre eclamtic and eclamptic is 4 to 12 percentage[78]. 30 percentage occur in postpartum period 70 percentage occurs in antepartum period in the third trimester and often associated in caucasian multiparous women aged about 25 years. Of the two thirds of women who are first diagnosed with HELLP syndrome antepartum, 10% will be identified before 27 weeks, 20% in pregnancies beyond 37 weeks, and the majority 70% occurring between 27 and 37 weeks gestation.

PATHOPHYSIOLOGY HELLP SYNDROME

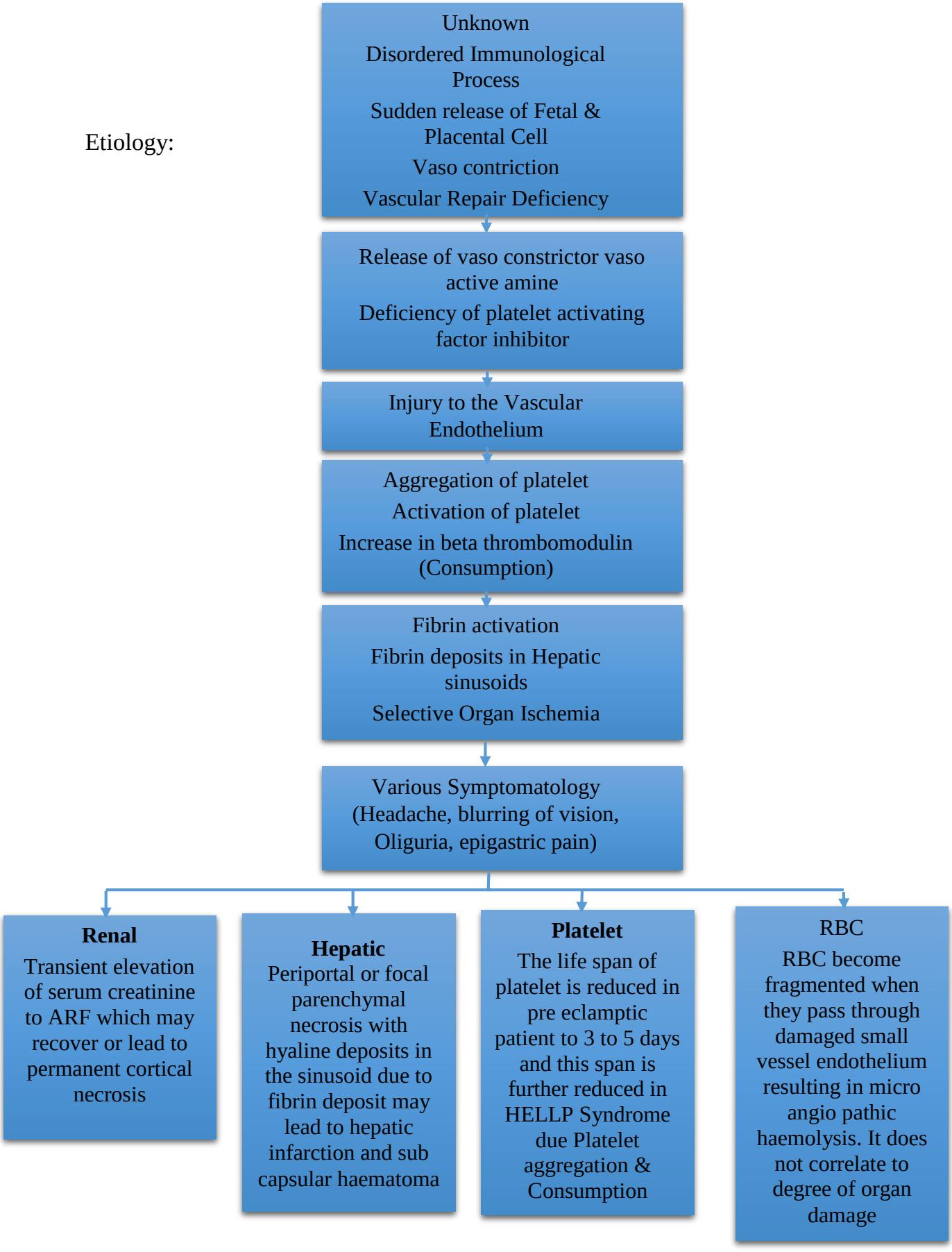
Etiology is unknown release of vaso constrictor vaso active amine, deficiency of platelet activating factor inhibitor, pathogenesis of HELLP syndrome is associated with factor V R 500 Q mutation (Brenner et al. 1996) Activated Protein C resistance resulting from mutation in coagulation factor V has recently emerged as the leading cause of thrombosis[44].

Patients with pure preeclampsia and HELLP Syndrome will have significantly higher level of serum C-erb B-2 encoded oncoprotein fragment P_{105} (Meden et al. 1997)[50]

Lower beta B-subunit Inhibin production in extra villous trophoblast cells in HELLP syndrome demonstrates that this subunit might have an important role in the pathogenesis of HELLP syndrome[52].

PATHOPHYSIOLOGY HELLP SYNDROME

Etiology:



Thrombocytopenia with eclampsia has been described at least since 1922 by Stancke. Because it is common, the platelet count is routinely measured in women with any form of gestational hypertension. The frequency and intensity of thrombocytopenia vary and are dependent on the severity and duration of the preeclampsia syndrome as well as the frequency with which platelet counts are performed (Heilmann and colleagues, 2007; Hupuczi and co-workers, 2007). Overt thrombocytopenia- defined by a platelet count < 100,000/μL- indicates severe disease. In general, the lower the platelet count, the higher the rates of maternal and fetal morbidity and mortality (Leduc and co-workers, 1992). In most cases, delivery is advisable because thrombocytopenia usually continues to worsen. After delivery, the platelet count may continue to decrease from the first day. It then usually increases progressively to reach a normal level within 3 to 5 days. With HELLP syndrome, the platelet count continues to raise after delivery. In some women whose platelet counts do not nadir until 48 to 72 hours, other causes of thrombocytotenia should be considered.

IMMUNOLOGY OF HELLP SYNDROME

Pre eclampsia has been considered for a number of years by many investigators to result at least in part, from disordered immunologic processes. The studies that support this belief, list the increased incidence in primigravida, the increased risk in pregnancies with an increased volume of trophoblastic tissue, pregnancy with a new partner, previous use of barrier contraceptive and pregnancy after oocyte donation. Since HELLP syndrome appears to be atypical form of preeclampsia, it too could result from disordered immunity.

Increased plasma levels of anaphylotoxins C3a and C5a have been demonstrated in patients with preeclampsia / HELLP syndrome. Depressions of both T-cell and B-cell potential and impaired monocyte handling of intracellular pathogens have been reported in pregnancies complicated by HELLP syndrome. This immune dysfunction preceded the laboratory diagnosis of preeclampsia by 7-14 days.

Haeger et al (1996) suggested that inflammatory mechanisms may participate in the pathophysiology of severe preeclampsia since increased release of Tumor necrosis factor alpha (TNFα) and interleukin 6 in women with HELLP Syndrome[41].

Dudley et al (1996) supported the hypothesis that the regulation of IL-12 production and metabolism is abnormal in women with preeclampsia and HELLP Syndrome, perhaps contributing to the immunologic alterations characteristics of these disorders[37].

Antiphospholipid antibodies may play a role in the pathogenesis of HELLP syndrome (Nagayama et al 1997)[45].

CLINICAL FEATURES

Patients with HELLP syndrome may present with various signs and symptoms, none of which are diagnostic and all of which be found in patients with severe preeclampsia-eclampsia without HELLP syndrome.

Prodromal Symptoms include (Portis et al., 1997)[46]

1. Weakness and fatigue (90%)
2. Right upper quadrant and/or epigastric pain (90%)

3. Nausea and Vomiting (50%)

4. Headache

5. Change in vision

6. Increased tendency to bleed from minor trauma

7. Jaundice

8. Diarrhoea

9. Shoulder or neck pain

SIGNS

1. Proteinuria $\geq$ 2+ (85%)

2. Hypertension Diastolic Blood Pressure $\geq$ 100 mm Hg (69%)

3. Significant weight gain with generalised edema (55%)

In Weinstein reports (1982/1985) nausea or vomiting and epigastric pain were the most common symptoms[6]. Sibai et al (1990) noted that the commonest symptom was epigastric and/or right upper quadrant pain[18]. Right upper quadrant or epigastric pain is used to assess whether the patient is at high risk for development of HELLP syndrome.

There is delay between the onset of symptoms and the fulfillment of diagnostic laboratory criteria (Koenen SV et al 2006)[55].

Severe hypertension (systolic blood pressure $\geq$ 160 mm Hg, diastolic blood pressure $\geq$ 110mm Hg) is not a constant or even a frequent finding in HELLP syndrome (Sibai et al 1986)[10].

Esan et al (1997) reported that HELLP syndrome can occur after a normal delivery in a woman whose blood pressure has remained normal throughout the antenatal period[49].

Donaldson (1978) reported that some may experience visual disturbances. Neurological affection can also result.

Jaundice is a rare complication and hyperbilirubinemia may result from a combination of hemolysis and liver cell necrosis. However it is unusual for icterus to be clinically apparent.

Maternal ascitis is frequently found at Caesarean delivery in 65% of patients with HELLP syndrome (Woods et al 1992)[22].

The risk of opportunistic infections may be increased in patients with HELLP syndrome, because of generalised (Both B&T Cell) immunosuppression and profound decrease in monocyte phagocytic and bactericidal activity (Cunningham et al. 1993)[25].

CLASSIFICATION

Classification system are of two types based on platelet count and LDH level which reflect the senility of disease process and rapidity of recovery from HELLP Syndrome

The Mississippi Triple Class System further classifies the syndrome based on the platelet count: Class I < 50 x ⟦10⟧^6/l and Class II – 50 x ⟦10⟧^6/l to 100 x ⟦10⟧^6/l and Class III- 100 x ⟦10⟧^6/l to 150 x ⟦10⟧^6/l. Classes I and II are associated with hemolysis (LDH > 600 U/l) and elevated AST(≥70U/l) concentration, while class III requires only LDH > 600 U/l and AST ≥40 U/l in

addition to the specific platelet count[61]. Class III HELLP syndrome is indicative of a clinically significant transition stage or a phase of the HELLP syndrome that may progress.

Mississippi 3-class (Martin et al)

THROMBOCYTOPENIA + HAEMOLYSIS + HEPATIC DYSFUNCTION

CLASS 1 : Platelets < 50,000 / µL
LDH > 600 IU/L
AST and/or ALT > 70 IU/L

CLASS 2 : Platelets > 50,000 - < 1,00,000/ µ L
LDH > 600 IU/L
AST and/or ALT > 70 IU/L

CLASS 3 : > 1,00,000 - < 1,50,000/ µ L
LDH > 600 IU/L

Tennessee (Memphis)
COMPLETE or TRUE
< 1,00,000/µ L Platelets
LDH > 600 IU/L
AST > 70 IU/L
INCOMPLETE or PARTIAL
Only one or two of above present + severe pre

Sibai proposed strict **diagnostic criteria** for HELLP syndrome

1. Intravascular hemolysis diagnosed by abnormal peripheral blood smear
2. Increased serum bilirubin ≥20.5 µmol/l or ≥1.2mg/100ml
3. Elevated LDH levels (>600 units/l)
4. Platelet count of < 100.000/microlitre

5. Serum AST (SGOT) levels > 70 IU/l
6. Low serum haptoglobin level
7. Drop in Hb% level unrelated to blood loss

OTHER CLASSIFICATIONS OF HELLP SYNDROME

	Platelet Count	AST	LDH
Sibai et al [11]	< 1,00,000/mm^3	≥ 70 IU/L	≥ 600 IU/L
Van Pampus[13]	< 1,00,000/mm^3	> 50 IU/L	>600 IU/L
Visser and Wallenburg[14]	< 1,00,000mm^3	> 30 IU/L	*

DIFFERENTIAL DIAGNOSIS FOR THE HELLP SYNDROME

- Different types of thrombocytopenia
  - Benign thrombocytopenia of pregnancy
  - Immunologic thrombocytopenia (ITP)
  - Thrombocytopenia due to folate deficiency
  - Systemic lupus erythematosus
  - Antiphospholipid syndrome (SLE)
  - Antiphospholipid syndrome (APS)
  - Thrombotic thrombocytopenic purpura (TTP)
  - Hemolytic uremic syndrome (HUS)
- Acute fatty liver of pregnancy
- Infectious and inflammatory disease
  - Urinary tract infections
  - Acute pancreatitis

    - Gastritis and gastric ulcers
    - Cholecystitis or cholangitis
    - Hepatitis
- Pyelonephritis and glomerulonephritis
- Hyperemesis gravidarum
- Kidney stones
- Hemorrhagic or septic shock
- Disseminated herpes simplex
- Acute renal failure with acute tubular necrosis
- Acute cocaine intoxication
- Pheochromocytoma
- Intrahepatic cholestasis

THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP) & HEMOLYTIC UREMIC SYNDROME (HUS)

- Renal abnormality is more severe
- Liver enzymes are usually normal
- Present during post partum period
- Severe anaemia is present

ACUTE FATTY LIVER OF PREGNANCY

- Liver enzymes are elevated but less than 1000
- Renal abnormality may be present
- Hypertension and proteinuria are absent
- Direct bilirubin is elevated

INTRA HEPATIC CHOLESTASIS

- Liver enzymes are elevated but less than 500
- No Renal abnormality
- Intense pruritis is present

VIRAL HEPATITIS

- Liver enzymes are elevated
- No Renal abnormality
- Abnormal serology

LABORATORY DIAGNOSIS

The diagnosis of HELLP Syndrome is based on laboratory values of Hemolysis liver dysfunction and low platelet in a patient with preeclampsia. Liver biopsy is considered as a gold stand for diagnosis but rarely needed because of hazardous laboratory work up includes complete blood count coagulation profile, serum creatinine, urinary protein, blood urea, serum uric acid, pheripheral blood smear and liver function test.

During disease progression thrombocytopenia occurs first and hemolysis occurs last. There is 50% drop of platelet in 24 hours. Thrombocytopenia is the principal and earliest coagulation abnormality that is present in all women with HELLP syndrome[32,33].

Laboratory abnormalities return to normal by fourth postpartum day, liver enzymes normalizes earlier than platelet count. Platelet count normalizes by 6-11 days[33].

Liver dysfunction is reflected by variably elevated serum concentration of aspartate amino transaminase (AST), alanine aminotransaminase (ALT) and LDH. Indirect levels of bilirubin usually are minimally elevated except in patients with advanced severe disease. Raised total Lactate dehydrogenase isoenzyme is usually reflected in elevations of isoenzymes 5 (LDH 5 liver). LDH1 and LDH2 are released by ruptured RBC's, but due to liver ischemia, the total LDH is elevated.

A peripheral blood smear often will have evidence of schistocytes, burr cells and helmet cells which reflect damaged erythrocytes. Increases in Lactic dehydrogenase (LDH) levels and decreases in serum Haptaglobin levels are sensitive early markers of HELLP syndrome.

A single A > G nucleotide substitution at position - 670 in the maternal but not neonatal TNFRSF6 gene coding for Fas is associated with a higher risk for HELLP syndrome (Sziller et al 2006)[57]. Significantly there will be a decreased expression of Pro apoptotic proteins BNip3 and Nix in the placenta of HELLP syndrome patients (Stepan H. et al 2005)[58]. O Rh-negative had HELLP syndrome associated with an increase in risk by a factor of 3.1 (Sezik M & Coworkers 2002)[59].

Iioka (1996) reported that increased level of Human Hepatocyte growth factor may be useful in the early detection of HELLP syndrome.

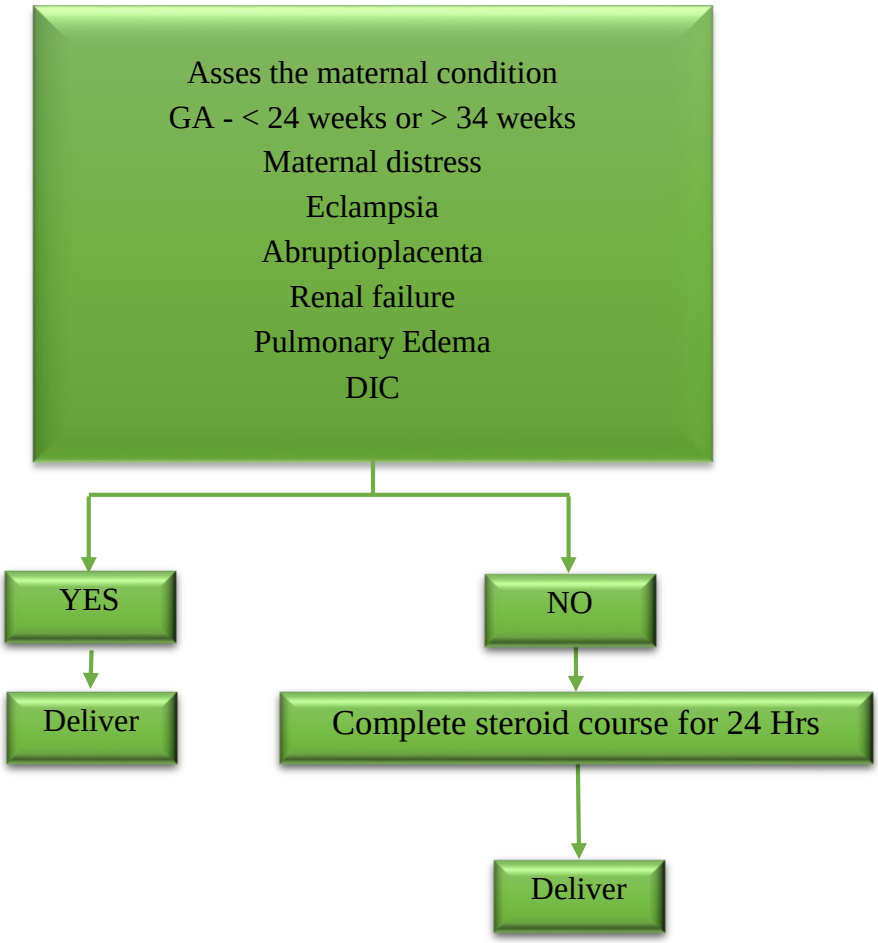
HELLP syndrome patients will have high plasma fibronectin and D-dimer values, lower Antithrombin III and protein C activity (Paternoster et al. 1995)[31].

Significant over expression of Serum Amyloid A (SAA) in HELLP syndrome patients that could function as markers for the HELLP syndrome (Heitner JC et al 2006)[56].

HELLP syndrome should be taken into account in the case of unexplained elevated levels of MSHCG and MSAFP in the second trimester especially in the rare event of combined elevation of both markers (Morssink-LP 1997)[48].

MANAGEMENT PROTOCOL FOR HELLP SYNDROME

- Refer to Tertiary Medical Centre.
- Admit in labor ward
- Do laboratory investigation.
- Prevent seizures with injection magnesium sulphate.
- Control of blood pressure with anti-hypertensive.
- Bed rest.
- Fluid and electrolyte correction.
- Hemotherapy with blood and blood products.



TREATMENT

The successful management of pregnancy complicated by HELLP Syndrome requires 12 step approach for better maternal and perinatal outcome.

1. ANTICIPATE AND CONFIRM THE DIAGNOSIS

The patient with evidence of preeclampsia with imminent signs and symptoms must be seriously evaluated to rule out HELLP syndrome. Once the diagnosis of HELLP Syndrome is suspected, appropriate laboratory testing is indicated. In its early phases, HELLP syndrome can exhibit only modest increases in LDH, AST and ALT levels and mild thrombocytopenia (Class 3). Timely diagnosis and early recognition and the institution of the appropriate therapy facilitates the best possible outcome for a mother and her unborn child.

Risk Factors

• **BIO CHEMICAL**	• **Imminent signs and symptoms**
Platelets < 50,000/ µ L	Epigastric pain
LDH > 1400 IU/L	Nausea
AST > 150 IU/L	Vomiting
ALT > 100 IU/L	Eclampsia
Uric Acid > 7.8 mg/dl	Severe Hypertension
Creatinine > 1.0 mg%	Placental Abruption

2. ASSESS THE MATERNAL CONDITION

Assess the maternal condition by clinically if there is maternal distress like Pulmonary Edema, DIC, MODS, etc., and if gestational age <24 weeks and more than 34 weeks and by all bio chemical investigations like CBC, urinalysis, serum creatinine, LDH, uric acid, indirect and total bilirubin levels, AST/ALT and coagulation profile. Serial assessments of the platelet count, LDH, and liver enzymes are reported usually every 12-24 hrs or more frequently if clinically indicated.

3. ASSESS THE FETAL CONDITION: DELIVER SOONER OR LATER

HELLP syndrome is an atypical variant of severe preeclampsia and, as such, the only definitive treatment is delivery and removal of chorionic villi

and the cytotoxic factor(s) that it produces. The timing of delivery depends on a number of factors, including the severity of the maternal condition, fetal condition and placental reserve, and the gestational age.

Fetal condition is assessed by fetal heart rate tracing, bio physical profile, or if GA is between 24 to 34 weeks there is a role for a course of steroids therapy for enhancing lung maturity.

A National Institute of Health consensus panel in 1994 recommended that all patients with pregnancies between 24 and 34 weeks gestation at risk for preterm delivery be considered candidates for corticosteroid therapy to enhance fetal lung maturation/neonatal pulmonary function even if delivery might not be postponed the ideal 24-48 hour period[66]. RCOG committee on Obstetrical practice has now adopted these recommendations.

4. CONTROL BLOOD PRESSURE (NICE Guide line 2010)

Antihypertensives used in HELLP Syndrome for acute control of blood pressure

- **Labetalol**
    - Starting doses 20mg IV followed by

      40mg and then 80mg at 10 – 15mins interval to a maximum doses of 220mg
- **Hydralazine**
    - Starting doses 5mg IV followed by

      5 – 10mg IV every 20mins to a maximum doses 20mg

- **Nifedepine**
    - Starting doses 10-20mg at 30mins interval maximum doses of 50mg

5. PREVENT SEIZURES WITH MAGNESIUM SULPHATE

It is recommended that all HELLP syndrome patients receive intravenously infused $MgSO_4$ given as a 4-6 gm intravenous bolus followed by a constant infusion rate of 2 mg/hr individualized to the patient. Continuation of the infusion 48 hrs or more into the puerperium occasionally is needed until evidence of recovery from HELLP syndrome is apparent.

6. MANAGE WITH FLUID AND ELECTROLYTES

Crystalloids and 5-25% of albumin

7. EXERCISE JUDICIOUS HEMOTHERAPY

- Packed cells
- Fresh Frozen plasma
- Platelet extract
- Cryo precipitate

8. MANAGE LABOUR AND DELIVERY

Treatment of the patient with HELLP syndrome includes careful assessment of the maternal and fetal status, with delivery effected soon thereafter.

This syndrome is not an indication for immediate delivery by caesarean section; vaginal delivery allowed when the GA is more than 32 weeks or during active labour or rupture of membranes. Induction and augmentation of

labour with prostaglandins or oxytocin can be used. Cervical status and inducebility are important considerations when determining the likelihood of successful vaginal delivery in patient with HELLP syndrome[54].

Caesarean delivery is recommended when GA is < 30 weeks with unfavourable cervix, absence of active labour, non-reassuring FHR, abnormal presentation, suspected abruption, severe oligohydraminos or IUGR are present.

Management protocol for the HELLP Syndrome patient requiring caesarean section delivery

Surgery to be scheduled for 6 hours from the beginning of high dose of steroids to stabilize the disease process, improve laboratory parameters, reduced the need for blood product transfusions. (Tompkin O Bien)[79]

1. General anesthesia
2. Ten units of platelets prior to surgery if platelet counts < 50,000/cu mm.
3. Vertical skin incision rather than a Pfannenstiel incision to minimise blood loss.

   Briggs et al concluded that for women with antepartum HELLP syndrome delivered by caesarean section, the frequency of wound complications is not influenced by type of skin incision or time of skin closure (Primary or delayed).
4. Spontaneous placental delivery rather than manual extraction at caesarean is associated with a decreased blood loss, and less postpartum endometritis.

5. In situ rather than exteriorization for uterine repair is associated with less uterine and adnexal trauma.
6. Bladder flap (vesicouterine peritoneum) should be left open.
7. Subfascial drain (24 to 48 hours).
8. Subcutaneous drain may be considered if the skin is approximated.
9. Subcutaneous tissues should be approximated with sutures as evidenced by randomized trials and meta analysis for skin closure at caesarean delivery.
10. Post operative transfusions as needed.
11. Intensive monitoring for 48 hrs postpartum.

ANAESTHESIA

- Maternal analgesia can be provided by intermittent infusion of butorphanol or meperidine with promethazine.
- Local infiltration 1% xylocaine for carefully controlled and skillfully executed non operative vaginal delivery.
- Epidural anesthesia and pudendal block is contraindicated.
- General anesthesia is the method of choice for caesarean delivery.
- When coagulopathy is evident before surgery, intradural anaesthesia (low doses of Bupivacaine and fentanyl) is a safe option provided hemodynamic stability is assured (Blasi et al 1997)[51].

9. OPTIMIZE PERINATAL CARE

Pernatal mortality is 10-20% and it is due to prematurity, IUGR, respiratory distress, low birth weight. These babies are instituted NICU care for better perinatal out come.

10. INTENSIVELY TREAT THE POSTPARTUM PATIENT

- The diagnostic criteria for HELLP syndrome may develop antepartum or postpartum. Sebai and associates revealed that 70% had evidence of the syndrome antepartum and 30% developed the criteria postpartum.
- In the postpartum group, the time of onset of the manifestations ranged from a few hours to 7 days, with the majority developing within 48 hours after delivery.
- HELLP syndrome may be diagnosed postpartum following 1 of 3 clinical scenarios:

1. Worsening of antepartum severe preeclampsia with delivery not yet altering the time course of the disease.
2. New onset of severe preeclampsia postpartum.
3. Rebound deterioration of a patient with antepartum HELLP syndrome after exposure to corticosteroid antepartum.

- Patients in this group are at increased risk for the development of pulmonary edema and acute renal failure.
- The goals of therapy postpartum differ compared with antepartum and are aimed solely at improving the maternal status.

- Management of seizure prophylaxis is similar to the antepartum patient with HELLP syndrome, including the need for $MgSO_4$.
- Hypertension control may be more aggressive, because there is no longer concern about compromising the uteroplacental circulation in the postpartum patient.
- Martin and coworkers recommended the trial of plasma exchange with fresh frozen plasma be considered in HELLP syndrome that persists past 72 hours from delivery and in which there is evidence of a life threatening microangiopathy[33].

11. REMAIN ALERT TO THE DEVELOPMENT OF MULTIPLE ORGAN SYSTEM FAILURE

12. COUNSEL ABOUT FUTURE PREGNANCIES

During the postpartum recovery period after a pregnancy with HELLP syndrome or at a later time remote from the index pregnancy, patients often ask for guidance about the recurrence risk for hypertension in general and HELLP syndrome specially in future pregnancies and 55% risk of recurrence if pervious delivery is less than 28 weeks.[29]

Currently there is no preventive therapy for recurrent HELLP syndrome. Birth weight and gestational age are the most important factors in predicting the course of a subsequent pregnancy.

There is higher incident of pre-eclampsia 20% in subsequent pregnancy in a patient who developed HELLP Syndrome (Facchinetti & Associates 2001). The overall incidence of recurrence is < 5%. The incidence of preterm delivery,

IUGR, abruption and IUD are higher in subsequent pregnancy. (Habli and associated 2009, Hnat and colleagues 2002).[68]

SUBSEQUENT PREGNANCY COMPLICATIONS: SIBAI ET AL (1995)

1.	Pre-eclampsia	19%
2.	Preterm delivery	21%
3.	IUGR	12%
4.	Abruptio placentae	2%
5.	Perinatal death	4%
6.	HELLP Syndrome	3-5%
7.	Chronic Hypertension	4%

Recurrence Risk for HELLP Syndrome or Preeclampsia

	HELLP Syndrome	**Pre eclampsia**
Sullivan et al	19 – 27%	23 - 43%
Lie 1998	-	13%
Van Pampus et al	2%	16%
Chames et al	6%	55%

Beinder et al 1996 reported that recurrence of HELLP Syndrome in four consecutive pregnancies in a patient.[40]

Infants born to pre eclamptic mothers who develop HELLP Syndrome have an increased need for resuscitation at delivery and a higher incidence of postnatal cardio pulmonary instability (Raval et al 1997).[47]

Sibai et al (1995) reported that there is no evidence that oral contraceptives should be contraindicated after HELLP syndrome.[35]

Steroids and the HELLP Syndrome

"DEXAMETHASONE RESCUE" for HELLP SYNDROME

ANTEPARTUM: 10 mg IV q 12 h

1. Whenever < 100,000/ µL Platelets
2. If Platelets 100,000 - 150,000/µL AND

   Eclampsia Severe Hypertension

   Epigastric pain "Fulminant Disease"

POSTPARTUM: 10 mg IV q 12 h x 2, then 5 mg IV q 12h x 2 individualised

1. Whenever antepartum steroids given to avoid rebound
2. Stop regimen after recovery evident (platelets > 100,000/ µL and LDH is trending downward and patients underlying preeclampsia / eclampsia is ameliorating

The mechanism of action is unknown but appears to alter the final steps in endothelial cell disruption.[75] Isler et al demonstrated intravenous dosing was superior to intramuscular dosing for several outcome variables including improving urine output and greater improvement in Laboratory values.

High-Dose Glucocorticoid Therapy for severe HELLP Syndrome

For most patients with HELLP syndrome, 10 mg intravenous dexamethasone every 6 hours for 2 doses followed by 6 mg intravenous dexamethosone every 6 hours for 2 additional doses.

For select patients at high risk, including those with profound thrombocytopenia (<20,000/ mm^3) or with central nervous system dysfunction (i.e. blindness, paralysis), 20 mg intravenous dexamethasone every 6 hours for upto 4 doses.

The duration of action of this medication is limited and patients may experience a worsening of their laboratory studies 48 to 72 hours after dosing with glucocorticoids. We term this as Rebound phenomenon. Steroid treatment, therefore, is not curative but may create a "Window of opportunity" for intervention before the maternal condition may again deteriorate. Because glucocorticoids do not appear to alter the underlying pathophysiology, delivery remains the only definitive therapy.[75]

Intravenous immunoglobulins might be an attractive alternative treatment in persistently severe HELLP Syndrome (O-Pourrat et al 1992).[20]

Another intervention to interrupt or ameliorate the clinical course of HELLP Syndrome includes the administration of nitric oxide.

MATERNAL MORBIDITY AND MORTALITY

Maternal morbidity and mortality is high in a patient with HELLP Syndrome particularly with complete HELLP Syndrome. Class-I and II have more complication than class-III.

In a review of 34 HELLP syndrome related mortalities, the authors discovered that the presenting symptom in 90% of patients who died was nausea-vomiting and right upper quadrant pain, the mean gestational age was 31 weeks, death occurred by a variety of pathologic processes, including sepsis, shock

hemorrhage, intra cerebral bleeding and cardiopulmonary failure. Approximately one in six (16%) maternal deaths was attributed to hepatic complications. A large percentage of maternal deaths attributed to central nervous system catastrophic events. The most important biochemical marker for maternal mortality is bilirubin levels. Maternal mortality was statistically higher in cases with jaundice (Demir SC et al 2006).[60]

Associated DIC is an important aggravating factor, often leading to deterioration of the maternal status, Van Dam et al (1989) suggested a semi quantitative DIC scoring system introduced by Hellgren et al (1984).[65] This DIC score is based on platelet count less than 100 x 10 9/7, Prothrombin time < 70%, antithrombin III activity < 80%, fibrin degradation products over 40 mg/L and fibrinogen < 300 mg/dl. Three or more pathologic tests were considered as manifest DIC and two as suspected DIC. DIC score may be a sensitive index for detecting deteriorating maternal condition in HELLP syndrome and its use could reduce maternal morbidity and mortality from DIC.

From Sibai et al (2003)[53]

1. Disseminated intravascular coagulopathy 21%
2. Abruptio placentae 16%
3. Acute renal failure 8%
4. Severe ascitis 8%
5. Pulmonary edema 6%
6. Pleural effusions 6%
7. Cerebral edema 1%

8. Retinal detachment 1%

9. Laryngeal edema 1%

10. Subcapsular liver hematoma 1%

11. Acute respiratory distress syndrome 1%

A rare but interesting complication of HELLP Syndrome is transient Diabetes Insipidus (Mabie & Sibai 16 1990).[11] It is characterised by a resistance to arginine vasopressin mediated by excessive vasopressinase. It is postulated that elevated circulating vasopressinase may result from impaired hepatic metabolism of the enzyme. The best prophylaxis against development of life threatening complications is early diagnosis and termination of pregnancy after stabilisation of the maternal condition, consisting of magnesium sulphate infusion, antihypertensive treatment with dihydralazine or calcium antagonists, steroids etc. As prophylaxis against postpartal worsening of HELLP syndrome, curettage of the uterus and continuation of the treatment with antihypertensives and dexamethasone have been recommended.

Maternal mortality is due to

- DIC
- MODS
- Pulmonary edema
- Intra cerebral haemorrage
- Post cesarean shock
- Hepatic rupture.

PERINATAL MORBIDITY AND MORTALITY

Perinatal morbidity and mortality in HELLP Syndrome is 10-20%. Mortality increase with early gestational age. The incidence of preterm delivery is 70%. Mortality is high due to preterm, low birth weight, IUGR, abruption.

Infant will have high rate of bronchopulmonary dysplasia, intra cerebral haemorrhage, necrotizing enterocolitis.

Perinatal mortality 33% (Sibai et al 1986, Eeltink et al 1993)[10] reported

- Small for gestational age 44%
- Perinatal asphyxia 21.6%
- Neonatal respiratory distress 43.2%
- Hyperbilirubinemia 44.7%
- Persistent ductus arteriosus 16.2%
- Thrombocytopenia 34%
- Hypoglycemia 16.2%

CAUSES FOR PERINATAL DEATH

1. Abruptio placentae
2. Intrauterine asphyxia
3. Prematurity

The combination of HELLP syndrome and eclampsia results in a greater number of preterm infants with lower birth weights and higher mortalities than eclampsia alone.

MATERIALS AND METHODS

METHOD OF COLLECTION OF DATA

Analysis of 100 cases of severe preeclampsia / eclampsia with HELLP syndrome and without HELLP syndorme during the year 2015 in Tirunelveli Medical College Hospital, Tirunelveli to determine the occurrence and course of HELLP Syndrome in order to make a timely intervention and to render optimal patient treatment, a better maternal and perinatal outcome.

The patients were divided into 2 groups

- Severe Preeclampsia / Eclampsia with HELLP syndrome (HELLP Group)
- Severe Preeclampsia / Eclampsia without HELLP syndrome(NON-HELLP Group)

History regarding age, parity, gestational age, menstrual history and pervious illness were noted. A thorough general and other systemic examination were done with obstetric examination.

The observations done were

1. Weight of the patient
2. Albuminuria
3. Blood Pressure
4. Haemoglobin
5. Platelet count
6. Peripheral smear
7. Serum Bilirubin estimation

8. SGOT estimation
9. SGPT estimation
10. BT
11. CT
12. Serum fibrinogen
13. Sr. LDH estimation
14. Blood urea and serum uric acid estimation
15. Serum Creatinine estimation
16. Fundus examination

Both groups were in admitted in labour ward kept under careful monitoring investigations like CBC, RFT, LFT, BT, CT, PT, aPTT, Fibrinogen were sent, and the intervention:

General Nursing Care, Electrolyte balance, monitoring urine output, Medical Management.

1. Antithypertensive drugs
2. Prophylactic Mgso4 for server preeclampsia

Obstetric Management

- Termination of pregnancy – based on Bishop score
- If Bishop Score is favourable, labour will be accelerated with oxytocin drip and ARM.
- If Bishop Score is unfavourable induction of labour with prostaglandins will be done.
- Caesarian section will be done for obstetric indications.

The women and newborn will be monitored for 1 week in the post partum period.

Outcome will be measured in terms of age of the mother, parity, socioeconomic status, antenatal care status, duration of gestation BP and degree of proteinuria, delivery interval, mode of delivery, perintal outcome and maternal outcome. Data obtained will be tabulated and analysed systematically.

According to NICE guidelines severe preeclampsia is managed conservatively until 34 weeks and delivery is recommended after a course of Corticosteroids after 34 weeks.

Delivery is recommended in a case of severe preeclampsia before 34 weeks after completion of steroids if severe hypertension is develops refractory to treatment and for maternal and fetal indication.

Source of data

Present clinical study will be carried on 100 cases of severe preeclampsia and eclampsia above 28 weeks of gestational age with HELLP syndrome admitted in Medical college Hospital, Tirunelveli for a period of 6 month from Feb 2015 to July 2015. These patient will be followed up prospectively till delivery.

Sample size: 100 cases

INCLUSION CRITERIA

All pregnant women above 28 weeks of gestational age with severe preeclampsia / eclamosia with one or more of the following:

- Hemolysis detected by either Peripheral smear or Elevated Indirect bilirubin or Elevated LDH levels.
- Elevated liver enzymes.
- Decreased Platelet count < 100,000/cumm.

Exclusion criteria

1. Known case of Hepatic disease.
2. Known case of Hemolytic Anaemias.
3. Known case of Platelet disorders.
4. Chronic hypertension in Pregnancy.
5. Chronic renal Diseases.
6. Placenta praevia.
7. Acute Fatty liver of Pregnancy.

Statistical analysis required

This prospective study requires Chi Square test for analysis.

PERIPHERAL SMEAR

Spreading the Film

Properly spread film is essential to accurate work.

1. The slides and cover glasses must be perfectly clean
2. Drop of blood must not be too large
3. The work must be done quickly

The blood is obtained from the finger tip. Take a small drop of blood on a clear slide about 3/4 inch from the end taking care that the slide does not touch the skin. Place the end of a second slide against the surface of the first an

angle of 30-40^{o} and draw it against the drop of blood, push the spreader slide back along the other. The blood will follow and then smears should be made. The film may be allowed to dry in the air. Leishman's stain is added, and after 2 mts double the quantity of distilled water are added to the stain and waited for 7-10 mts. Then the stain is washed in the tap water and slide dried in air and viewed under oil immersion.

The mature red corpuscle appears greenish yellow in unstained preparations and is roughly circular in shape and seen on edge as biconcave disc. Cells with reduced concentration of heamoglobin are called hypochromic which may be so extensive that only a narrow rim of haemoglobin is left around the periphery. There cells are called pessary cells.

Spherocyte are small darkly staining cells. Burr cells are mature red cells which possess one or more spiky projecting on their periphery seen in microangiopathic haemolytic anaemia.

SERUM BILIRUBIN ESTIMATION

Methods of detecting and estimating Bilirubin in serum are based on the formation of the purple compound when bilirubin reacts with the diazo reagent introduced by vanden Berg.

Reagents

1. Absolute method
2. Diazo reagent prepared freshly by adding 0.3 ml of solution B to 10 ml of solution A.

a. Solution A

Dissolve 1 gm of sulphuric acid in 15 ml concentrated HC1 and make it to i liter with distilled water.

b. Solution B

- 0.5% sodium nitrite in water prepared at frequent intervals.
- Prepare a solution containing 10 mgm/100ml of chloroform. It may be necessary to reflex the mixture gently to dissolve the bilirubin.

Technique

Wash 0.2 ml of serum into 5.4 ml of 0.9% saline. For values above 15 mgms/0.1 ml serum in 5.5 ml water may be taken serum 0.2, 5.6 ml divided into 4 parts of 1.4 ml each.

	Total Bilirubin		Direct Bilirubin	
	Test	**Control**	**Test**	**Control**
Dilute Screen	1.4 ml	1.4 ml	1.4 ml	1.4 ml
UDBA	-	0.35 ml	-	0.35 ml
Diazo reagent	0.35	-	0.35	-
Water	-	-	1.75	1.75
Methanol	1.75	1.75	-	-

Let it stand for 10 mts read against water blank at 530mμ photoelectric colorimeter.

Standardization

Mg/bilirubin per 100 ml 0-10-20-30-40

MI Standard solution	**0.1**	**0.2**	**0.3**	**0.4**	**-**
MI Chloroform	2.8	2.7	2.6	2.5	2.4
Diazo reagent	0.7	0.7	0.7	0.7	0.7
Methanol	3.5	3.5	3.5	3.5	3.5

Bilirubin Normal Value Total <1mg%

LDH ESTIMATION

Sample

Although serum is preferred, plasma can be used only if heparin or EDTA is used as an anticoagulant.

Serum or plasma should be separated from the blood sample as early as possible.

Serum or plasma can be used stored at 2-8^{o}C for one week.

Reagents: The Ames Autopak LDH reagent kit

1. NADH

1A. Buffer

2. Pyruvate

Preparation of working solutions

Allow the reagents to attain the room temperature. The Ames Autopak LDH reagent kit contains three bottles/vials each of reagent 1 & 1A and one bottle of reagent 2.

Solution (1): Transfer a small amount of the contents of one bottle (1A) into one bottle

(1). Mix gently to dissolve and transfer the solution back to bottle (1A). Rinse bottle (1) with solution in bottle (1A).

Reagent (2): Ready for use

Preparation of Daily working solutions

Allow the solution (1) and reagent (2) to attain the room temperature.

The daily working solution should be prepared fresh according to the need. Mix 10 volumes of solution (1) with 1 volume of reagent (2) to obtain daily working solution.

Storage and stability of the Reagents

Expiry date of reagents stored at 2-8^{o}C is indicated on the box label. Solution (1) is stable for six weeks at 2-8^{o}C Reagent (2) is stable at 2-8^{o}C until the end of expiry date on the table.

Procedure

The samples and the daily working solution should be brought to room temperature prior to use. The general system parameters are

Reaction Type	:	Kinetic
Wavelength	:	340mm
Flow cell temperature	:	37^{o}C
Delay time	:	60 Sec
No. of Readings	:	4

Interval	:	30 Sec
Sample Volume	:	30 µl
Reagent Volume	:	1.0 ml
Pathlength	:	1 cm
Factor	:	5520
Zero setting with	:	Distilled water

Procedure limitations

Plasma can be used only if heparin or EDTA is used as an anticoagulant Citrate and oxalate interfere with the test and hence should not be used.

Do not use haemolysed samples as haemolysis may give falsely elevated results.

The method is linear upto 1000 IU/L. For higher values, dilute the sample suitably with 0.9% saline and repeat the assay. Apply proper dilution factor to calculate the final results.

Normal Values

Upto 600 IU/L (37ºC)

SGOT ESTIMATION

Sample Collection, Storage and Stability

Although serum is preferred, plasma can be used. Anticoagulants such as heparin and EDTA can be used. Blood samples may be collected any time, although morning samples are preferred.

Samples with any visible hemolysis are not acceptable. Samples are stable for a

week at 2-8^0 C and for one month at 10^{o}C samples should be brought to room temperature prior to use.

Reagents

1. Aspartate/Buffer

1A. NADH/MDH/LDH

2. Alpha-Ketoglutarate

Preparation of working solutions

Allow the reagents to attain the room temperature.

Solution (1): Quantitatively transfer the contents of Via 1A into bottle 1. Mix until completely dissolved.

Solution (2): Dissolve the contents of bottle 2 with 14 ml of distilled water.

Preparation of daily working solutions

Allow the solutions (1) and (2) to attain the room temperature.

The daily working solution should be prepared freshly according to the need in the proportions given below: **Solution (1):** 3.0 ml

Solution (2): 0.3 ml

Mix thoroughly, use within 8 hours of preparation - store at 2-8^{0}C.

Storage and stability of Reagents

Expiry date of reagents stored at 2-8^{0}C are

Solution (1):for 3 months

Solution (2):for 4 months

Procedure

The samples and the daily working solution should be at the room temperature prior to use. The following general system parameter are used.

Reaction Type : Kinetic

Wavelength : 340 mm

Flowcell Temp : 37^0C

Delay Time : 60 Sec

No. of Readings : 4

Interval : 30 Sec

Sample Volume : 100 ml

Reagent Volume : 1.0 ml

Pathlength : 1 cm

Factor : 1749

Zero setting with : Distilled water

Procedure limitations

- Haemolysis of sample can produce a significant positive error as red cells contain large amounts of GOT (AST).
- Samples with very high GOT activity cause an excessive consumption of NADH, resulting in a very low initial absorbance. When this occurs, the assay should be repeated with a diluted sample.

- Linearity of the method is upto 400 IU/L for higher values, dilute the sample suitably with 0.9% saline and repeat the assay. Apply proper dilution factor to calculate the final results.

Normal Values

Upto 40 IU/L (37ºC)

SGPT ESTIMATION

Sample

Although serum is preferred, plasma can also be used with anticoagulants such as heparin or EDTA.

Serum or plasma GPT determination should be carried out on the same day as far as possible.

Samples are stable for about 3 days when stored tightly capped at 2-8^{0}C or for two weeks at – 10ºC.

Avoid using haemolysed or grossly contaminated samples. The samples should be brought to room temperature prior to use.

Reagents

1. Alanine / Buffer 1A. NADH/LDH
2. Alpha - Ketoglutarate

Preparation of working solutions, storage and stability of reagents, procedure & procedure limitation.

Same as that for SGOT.

Normal values

Upto 40 IU/L (37ºC)

METHOD OF COUNTING PLATELETS

Rapid work is necessary in order to prevent clumping of the thrombocytes. Rees & Eker diluting fluid drawn upto 1 mark in the red pipette. Blood from a freely bleeding puncture is drawn exactly to the 0.5 mark and finally the diluting fluid is quickly drawn to the 101 mark. This gives a blood dilution of 1:200. The blood and diluting fluids are immediately mixed by shaking for about 2 mts. The counting chamber is filled at once and 10 mts are allowed for the corpuscles to settle before counting is begun. The count is made with high power dry objective and with the 10x ocular in the manner described for counting erythrocytes. A central count of the thrombocytes should always be made at the same time with the same diluting fluid and exactly the same technique. Platelet count were also obtained from peripheral smear.

Platelets are disc shaped being 2-4 μm in diameter and 0.5 to 1mm in thickness. They have no nucleus and their cytoplasm has many azurophil granules which in blood films tend to be concentrated in the middle. The precursor for the platelet is megakaryocytes. The platelets are formed by fragmentation and detachment of delicate processes from cytoplasm of megakaryocyte. The normal platelet count varies from 150000 to 400000/mm^3. A count below 100000/mm^3 can be taken as thrombocytopenia.

STATISTICAL ANALYSIS:

The group with HELLP syndrome and the group without HELLP syndrome have been described and interpreted according to their demographic, social, physiological biochemical and obstetric characteristics. The maternal outcome of and perinatal outcome were analyzed and interpreted as follows. In the above description and analysis, the measurable variables were compared between the two by mean and standard deviations. The student t test was applied to infer the significance of difference between the means. The categorical variables were compared between them by Chi-square test (χ^2). The statistical analysis and interpretations have been performed by the statistical software IBM SPSS-sdtatistics-20. The P-value less than 0.05 ($P<0.05$) was treated as statistically significant in two tail test.

RESULTS:

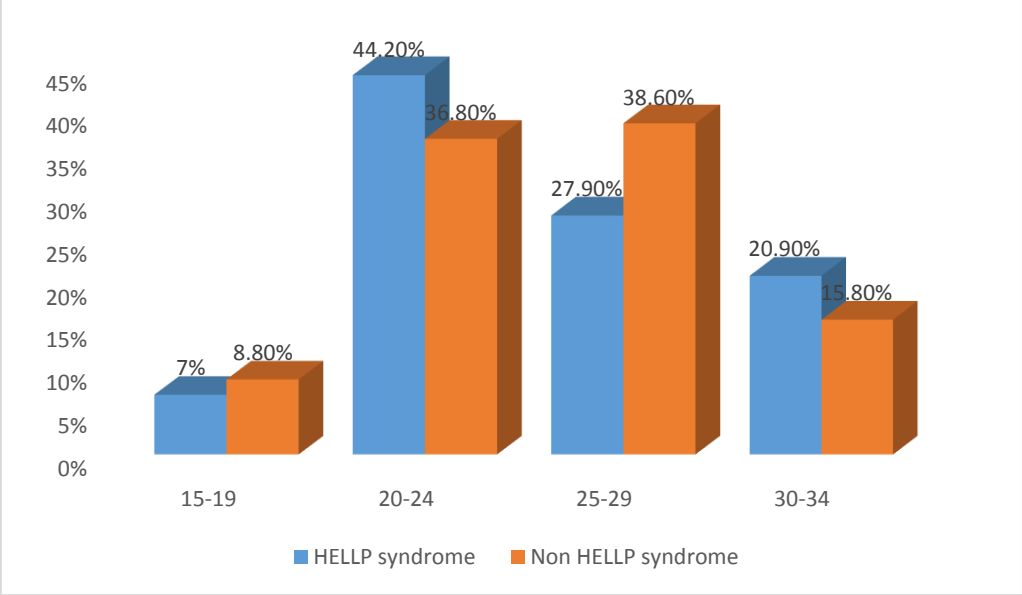
The group with HELLP syndrome and group without HELLP syndrome were described and compared between the demographic, economic, physiological, biochemical and obstetrics characteristics. Among the selected 100 mothers, 43 mothers were having HELLP syndrome and the remaining 57 did not have the HELLP syndrome.

DEMOGRAPHIC CHARACTERISTICS

The study subjects were described according to their age and compared between them.

Table: 1. Percentage distribution of study subjects according to their age:

Age group	HELLP syndrome		No HELLP syndrome		Total	
	No	%	No	%	No	%
15-19	3	7.0	5	8.8	8	8.0
20-24	19	44.2	21	36.8	40	40.0
25-29	12	27.9	22	38.6	34	34.0
30-34	9	20.9	9	15.8	18	18.0
Total	43	100.0	57	100.0	43	100.0
Mean ±SD	25.2 ±4.3		25.0±4.1		25.1±4.2	
't'	t=0.260				Age range = 18-34	
Significance	P>0.05					



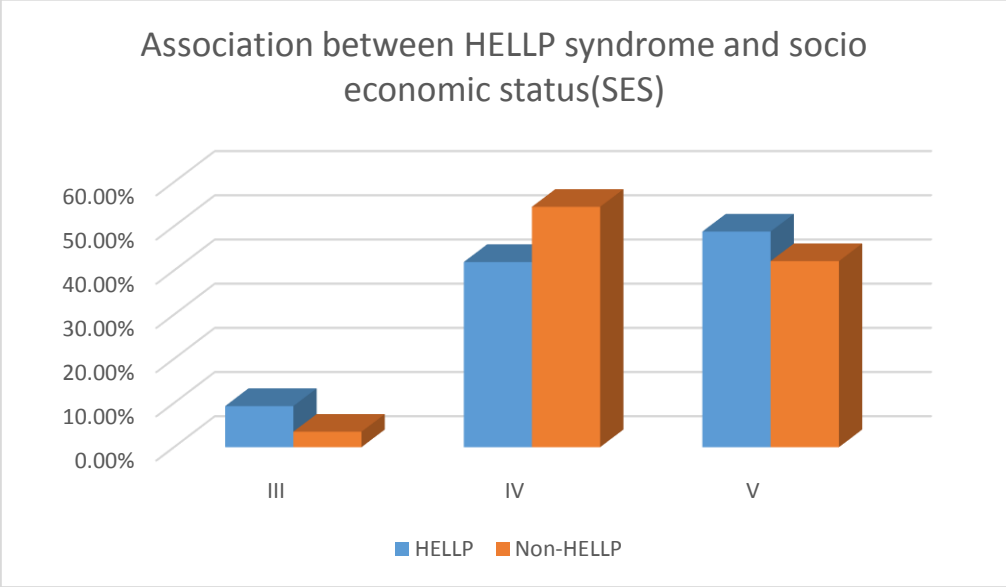
44.2% of HELLP group belong to the age group of 20-24 years, and 38.6% of Non-HELLP group belong to the age group of 25-29 years. Hence the difference between the two group were not statistically significant (P>0.05).

SOCIO ECONOMIC CHARACTERISTICS

The socio economic characteristics of the HELLP syndrome subjects were studied to identify the association between socioeconomic status and HELLP syndrome.

Table: 2. Association between HELLP syndrome and socio economic status(SES):

SES	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
III	4	9.3	2	3.5	6	6.0			
IV	18	41.9	31	54.4	49	49.0	2.043	2	P>0.05
V	21	48.8	24	42.1	45	45.0			
Total	43	100.0	57	100.0	100	100.0			



48.8% of HELLP group belongs to 5th socio economic status and 54.4% of Non-HELLP group belong to 4th socio economic status. But the SES of study does not show any significant association between both groups (P>0.05).

PHYSIOLOGICAL CHARACTERISTICS

The study subjects namely HELLP syndrome cases were studied according to their physiological characteristics namely weights, Systolic BP and Diastolic BP. The same was compared with non HELLP syndrome cases.

Table: 3. Comparison of Weight, SBP and DBP between HELLP and non HELLP:

Variable	HELLP, n=43		Non HELLP, n=57		Difference b/w means	't'	df	Significance
	Mean	SD	Mean	SD				
Weight	56.7	6.5	56.6	5.0	0.1	0.027	98	P>0.05
SBP	162.9	19.8	160.0	14.9	2.9	0.844	98	P>0.05
DBP	112.4	11.9	112.3	10.3	0.1	0.020	98	P>0.05

The mean SBP of HELLP and non HELLP group was 162.9±19.8 and 160.0±14.9 mm/hg respectively. The mean DBP of HELLP and non HELLP group was 112.4±11.9 and 112.3±10.3 mm/hg respectively. The difference of means between both groups was not statistically significant (P>0.05).

BIOCHEMICAL CHARACTERISTICS

The biochemical variables like blood urea, serum creatinine, Hemoglobin, Serum uric acid, Serum bilirubin-Total, Serum bilirubin-Direct, and Serum bilirubin-Indirect have been studied and compared between the HELLP and non HELLP mothers.

Table: 4. Comparison of biochemical variables between HELLP and non HELLP:

Variable	**HELLP, n=43**		**Non HELLP, n=57**		**Difference b/w means**	**'t'**	**df**	**Significance**
	Mean	SD	Mean	SD				
BU	25.3	10.6	24.2	8.4	1.1	0.578	98	P>0.05
SC	1.2	1.1	1.1	1.7	0.1	0.246	98	P>0.05
UA	7.2	1.6	6.7	1.8	0.5	1.479	98	P>0.05

The mean blood urea (BU) of HELLP and Non HELLP syndrome mothers were 25.3±10.6 and 24.2±8.4 respectively. The difference between them was not statistically significant (P>0.05). The mean serum creatinine of both groups was 1.2±1.1 and 1.1±1.7 respectively. The difference of means was not statistically significant (P>0.05). The mean uric acid (UA) of HELLP group was 7.2±1.6 and the same of non HELLP group was 6.7±1.8. The difference was also not statistically significant (P>0.05).

Table: 4.1. Comparison of biochemical variables between HELLP and non HELLP

Variable	HELLP, n=43		Non HELLP, n=57		Difference b/w means	't'	df	`Significance
	Mean	SD	Mean	SD				
HB	7.8	2.5	10.8	8.4	3.0	2.223	98	P<0.05

The mean Hemoglobin level of HELLP syndrome group was 7.8±2.5 and non HELLP group was 10.8±8.4. The mean difference between the two groups was statistically significant (P<0.05). It shows there is drop in Hemoglobin level in HELLP than Non-HELLP group.

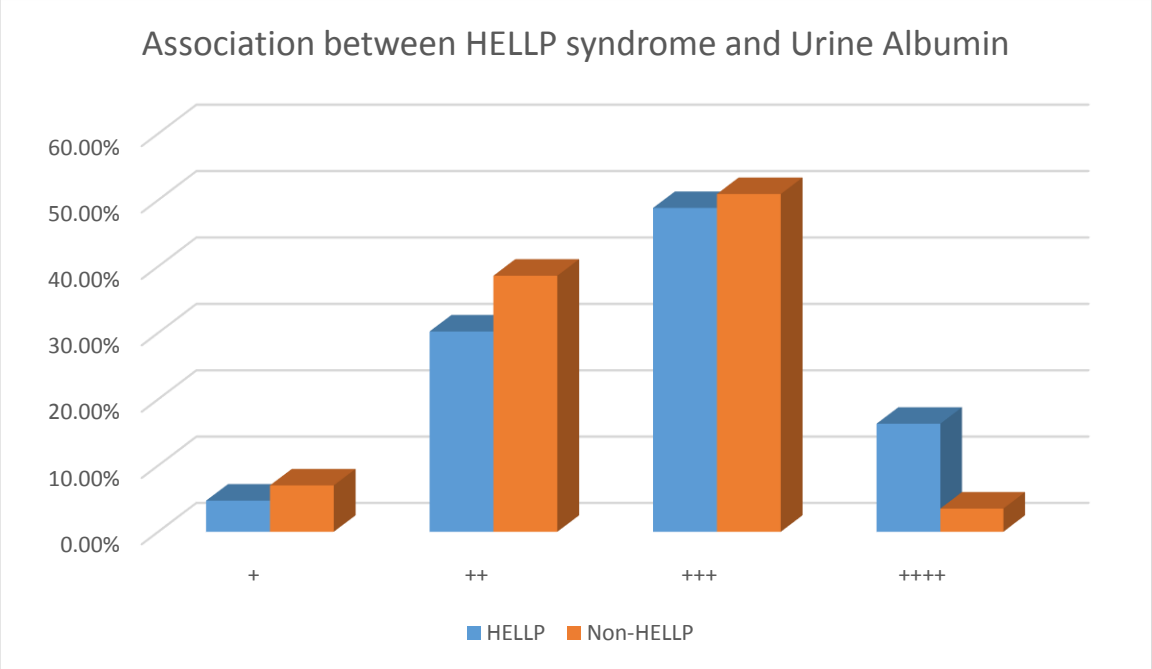
Table: 4.2. Comparison of biochemical variables between HELLP and non HELLP

Variable	HELLP, n=43		Non HELLP, n=57		Difference b/w means	't'	df	`Significance
	Mean	SD	Mean	SD				
S B-T	1.9	1.5	0.8	2.3	1.1	5.156	98	P<0.001
S B-D	0.8	0.6	0.5	0.2	0.3	3.648	98	P<0.001
S B-I	1.1	1.0	0.3	0.1	0.8	5.475	98	P<0.001

The mean serum biluribin Total, Direct and Indirect of HELLP group was 1.9±1.5, 0.8±0.6 and 1.1±1.0 respectively. Similarly, the mean serum biluribin Total, Direct and Indirect of non HELLP group was 0.8±2.3, 0.5±0.2 and 0.3±0.1 respectively. The differences between the groups were statistically very highly significant (P<0.001). The serum indirect biluribin was more among HELLP syndrome.

Table: 5. Association between HELLP syndrome and Urine Albumin:

UA	**HELLP**		**Non-HELLP**		**Total**		**χ^2**	**df**	**Significance**
	No	%	No	%	No	%			
+	2	4.7	4	7.0	6	6.0			
++	13	30.2	22	38.6	35	35.0	5.180	3	P>0.05
+++	21	48.8	29	50.9	50	50.0			
++++	7	16.3	2	3.5	9	9.0			
Total	43	100.0	57	100.0	100	100.0			



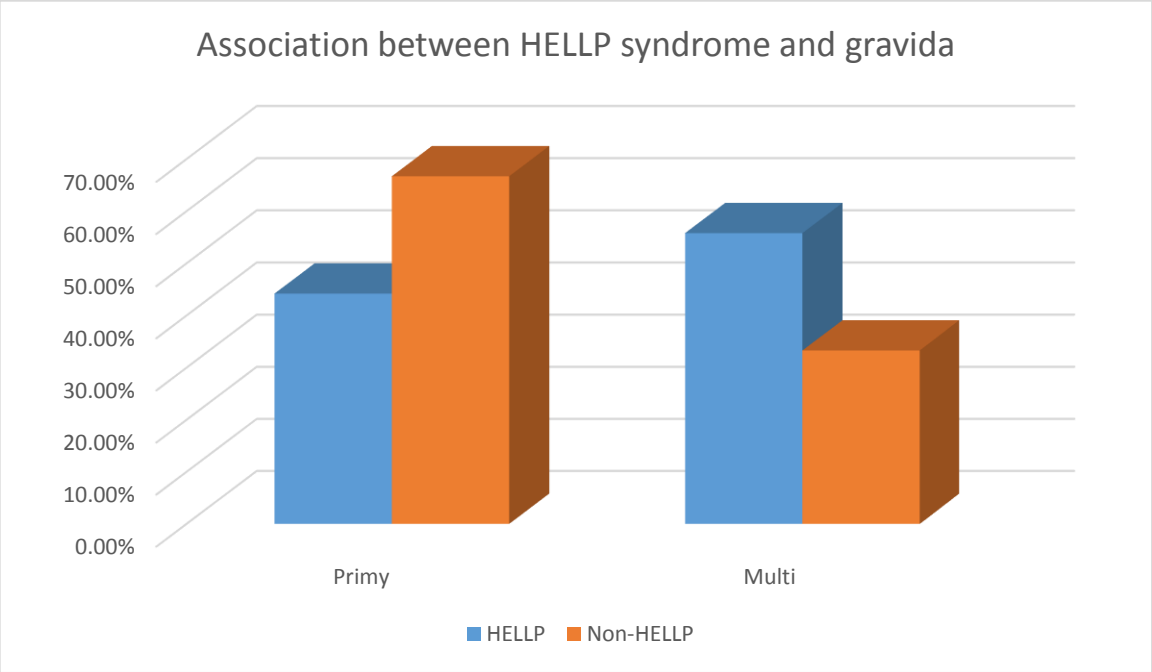
48.8% among the HELLP group has more than 3+ proteinuria and 50.9% among the Non HELLP has proteinuria. The above table show the there is significant proteinuria in both HELLP and Non-HELLP group and they are not statically significant p>0.05.

OBSTETRIC CHARACTERS

The HELLP mothers were described according to their obstetric characters like gravida, gestational age and Fundus,

Table: 6. Association between HELLP syndrome and gravida:

Gravida	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Primi	19	44.2	38	66.7	57	57.0	5.054	1	P<0.05
Multi	24	55.8	19	33.3	43	43.0			
Total	43	100.0	57	100.0	100	100.0			



55.8% of pregnancy in HELLP group were multi gravida and 57% of pregnancy in Non-HELLP group were primi gravida. According to the above table HELLP syndrome shows higher incidence in multipara.

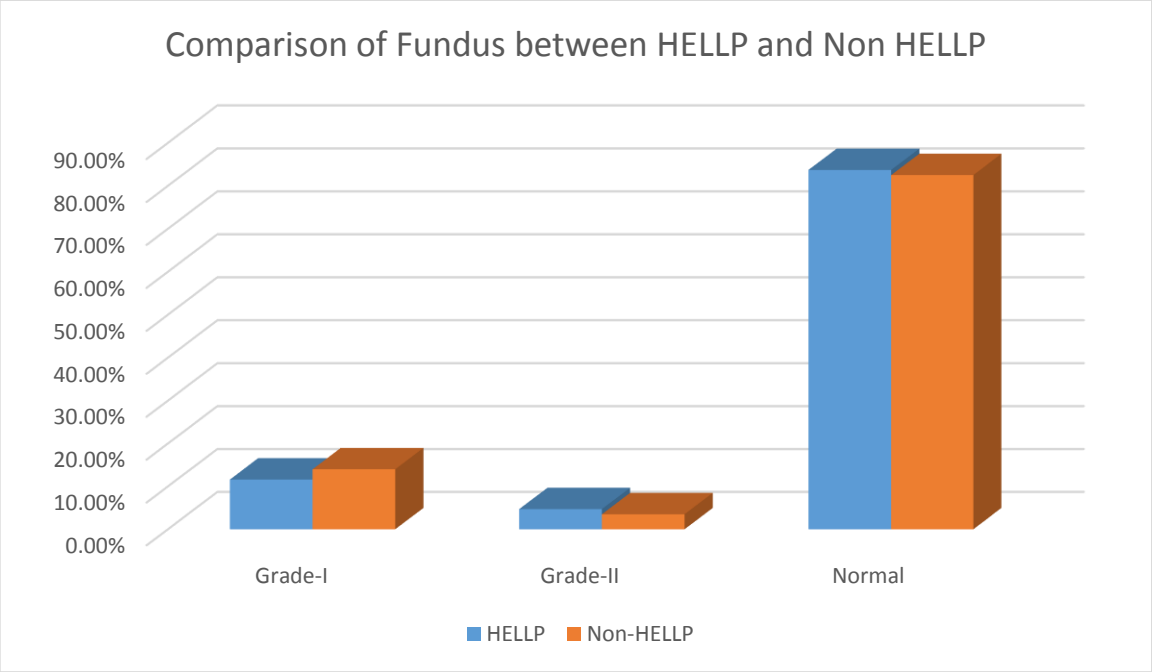
Table: 7. Comparison of Gestational age of HELLP with non HELLP.

HELLP		**Non-HELLP**		**Difference**	**'t'**	**df**	**Significance**
Mean	SD	Mean	SD	**b/w means**			
33.2	3.5	34.8	3.1	1.6	2.424	98	P>0.05

The mean gestational age of HELLP group was 33.2±3.5 weeks and the same of the non HELLP group was 34.8±3.1 weeks. The difference between them was not statistically significant (P>0.05). According to the above table HELLP syndrome is more common around 33 -34weeks of gestation.

Table: 8. Comparison of Fundus between HELLP and Non HELLP:

Fundus	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%	0.194	2	P>0.05
Grade-I	5	11.6	8	14.0	13	13.0			
Grade-II	2	4.7	2	3.5	4	4.0			
Normal	36	83.7	47	82.5	83	83.0			
Total	43	100.0	57	100.0	100	100.0			



In both groups, the fundus was normal as 83.7% and 82.5% respectively. There was no significant association between them (P>0.05). 16.3% cases had fundus changes in HELLP syndrome.

HELLP criterion characteristics:

The HELLP criterion measurements namely SGOT, SGPT, LDH and platelet have been compared between the HELLP and non HELLP mothers.

Table: 9. Comparison of SGOT, SGPT, LDH, Platelet between HELLP and non HELLP:

Variable	HELLP, n=43		Non HELLP, n=57		Difference b/w means	't'	df	Significance
	Mean	SD	Mean	SD				
SGOT	111.6	59.2	35.9	24.0	75.7	8.717	98	P<0.001
SGPT	122.6	101.0	35.2	25.4	87.4	6.282	98	P<0.001
LDH	1452.6	1136.5	246.9	171.9	1205.7	7.902	98	P<0.001
Platelet	69113.5	26919.7	194315	60999	125202.3	12.557	98	P<0.001

The mean SGOT, SGPT, LDH and Platelet of HELLP group were 111.6 ± 59.2, 122.6±101.0, 1452.6 ±1136.5 and 69113.5±26919.7 respectively. The same of the non HELLP group were 35.9±24.0, 35.2±25.4, 246.9±171.9and 194315±60999 respectively. The differences between the measures of the groups were statistically very highly significant (P<0.001). Isolated elevation of liver

enzymes are found in Non-HELLP group but there is significant elevation of liver enzymes in HELLP syndrome.

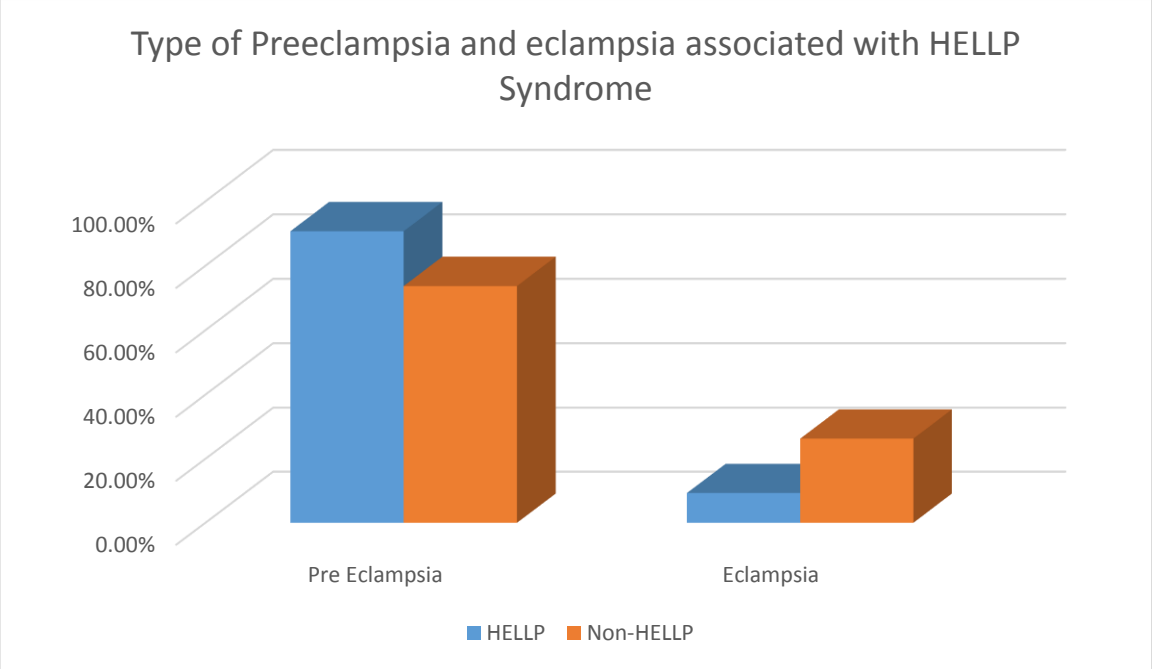
Table: 9.1. Platelet

Platelet	HELLP syndrome	
	No. of Cases	Percentage
Class-I < 50000	13	30.2%
Class-II 50000 – 1 lakh	24	55.8%
Class-III 1 lakh - 1.5 lakh	6	14%

According to the above table 30.2% of HELLP syndrome belonged to class-I, 55.8% belong to class-II, and 14% belong to class-III.

Table: 10.Association of Pre-Eclampsia or Eclampsia associated with HELLP mothers:

TYPE	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Pre Eclampsia	39	90.7	42	73.7	81	81.0	4.610	1	P<0.05
Eclampsia	4	9.3	15	26.3	19	19.0			
Total	43	100.0	57	100.0	100	100.0			



From the above table -9, 90.7% of Pre-Eclampsia was associated with HELLP syndrome. The association between them was statistically significant (P<0.05). HELLP syndrome is more come in severe preeclamptic patients.

Table: 11. Symptoms classified between the HELLP and non HELLP.

Symptoms	HELLP		Non HELLP		Total	
	No	%	No	%	No	%
Nil	8	18.6	22	38.6	30	30.0
↓UO	7	16.3	3	5.3	10	10.0
B/H	1	2.3	0	0.0	1	1.0
C	7	16.2	14	24.6	21	21.0
H	17	39.6	13	22.9	30	30.0
V	3	7	5	8.8	8	8.0
Total	43	100.0	57	100.0	100	100.0

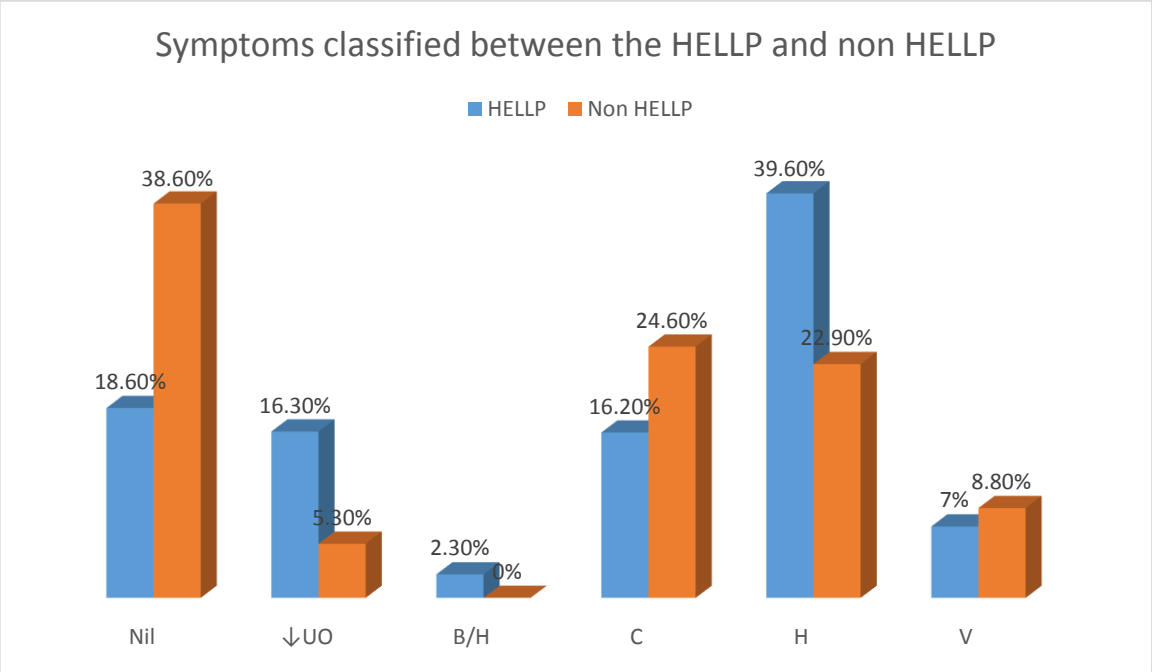
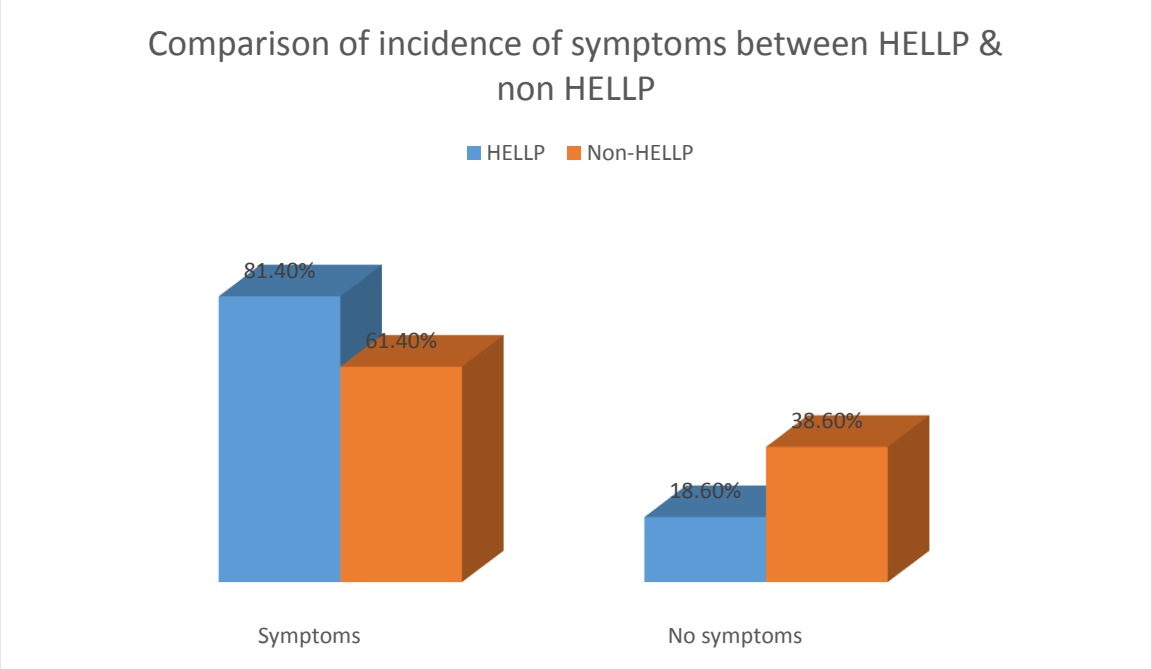


Table: 12. Comparison of incidence of symptoms between HELLP & non HELLP:

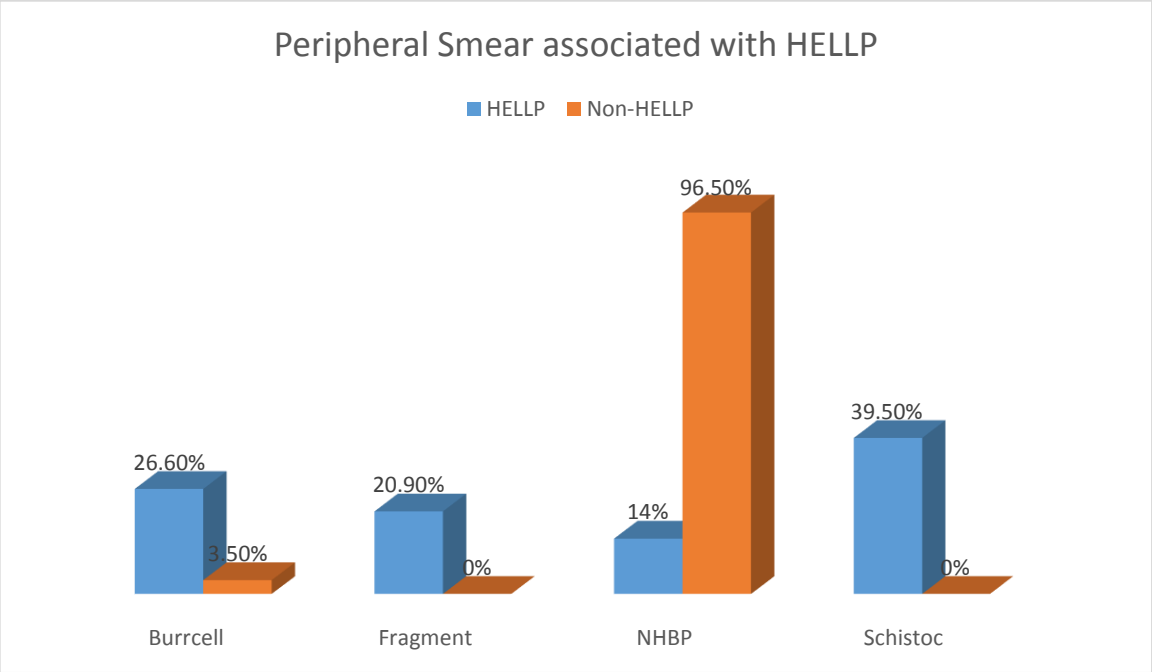
TYPE	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Symptoms	35	81.4	35	61.4	70	70.0	4.665	1	P<0.05
No symptoms	8	18.6	22	38.6	30	30.0			
Total	43	100.0	57	100.0	100	100.0			



81.4% of HELLP group patient present with symptoms and 61.4% of Non-HELLP group patient present with symptoms. The association of symptoms with HELLP is statistically significant p<0.05.

Table: 13. Peripheral Smear associated with HELLP:

Peripheral Smear	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Burrcell	11	26.6	2	3.5	13	13.0	71.023	1	P<0.001
Fragment	9	20.9	0	0.0	9	9.0			
NHBP	6	14.0	55	96.5	61	61.0			
Schistoc	17	39.5	0	0.0	17	17.0			
Total	43	100.0	57	100.0	100	100.0			



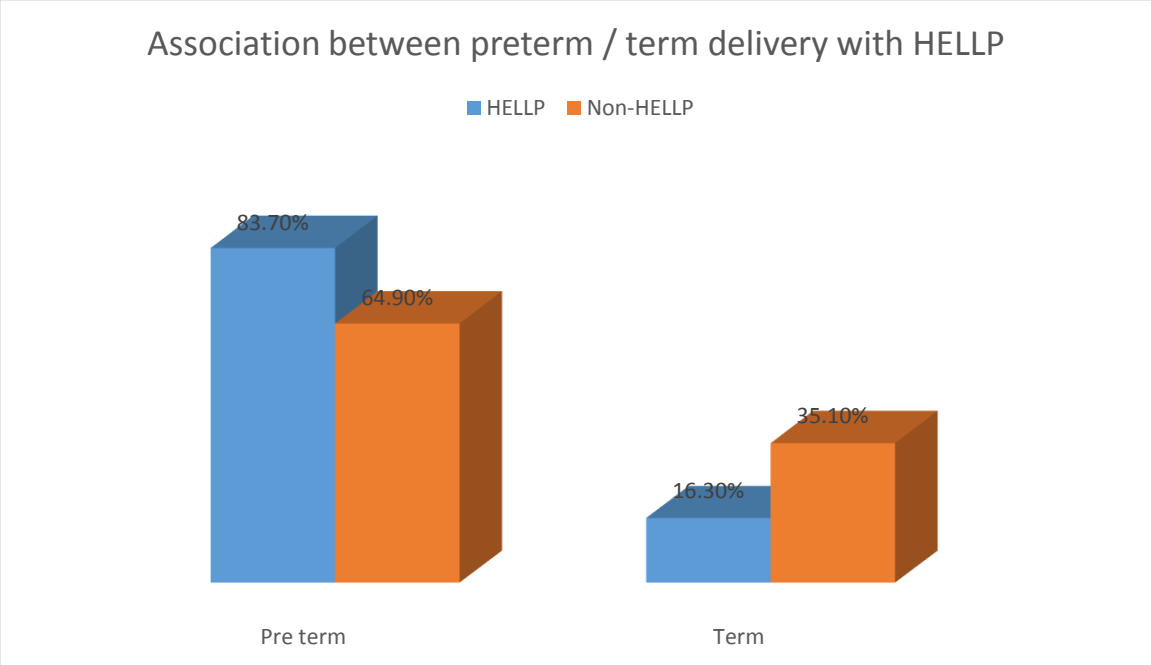
86% of HELLP group shows micro angiopathic haemolysis and only 3.5% of Non-HELLP group shows haemolysis. Hence the Peripheral smear results such as Burrcell, Fragmented cells and Schistocytes were very strongly associated with HELLP groups ($P<0.001$).

MATERNAL OUTCOME

The maternal outcome of HELLP group was studied in respect of their Term of delivery, mode of delivery, blood transfusion, complications and condition of mothers.

Table: 14. Association between preterm / term delivery with HELLP.

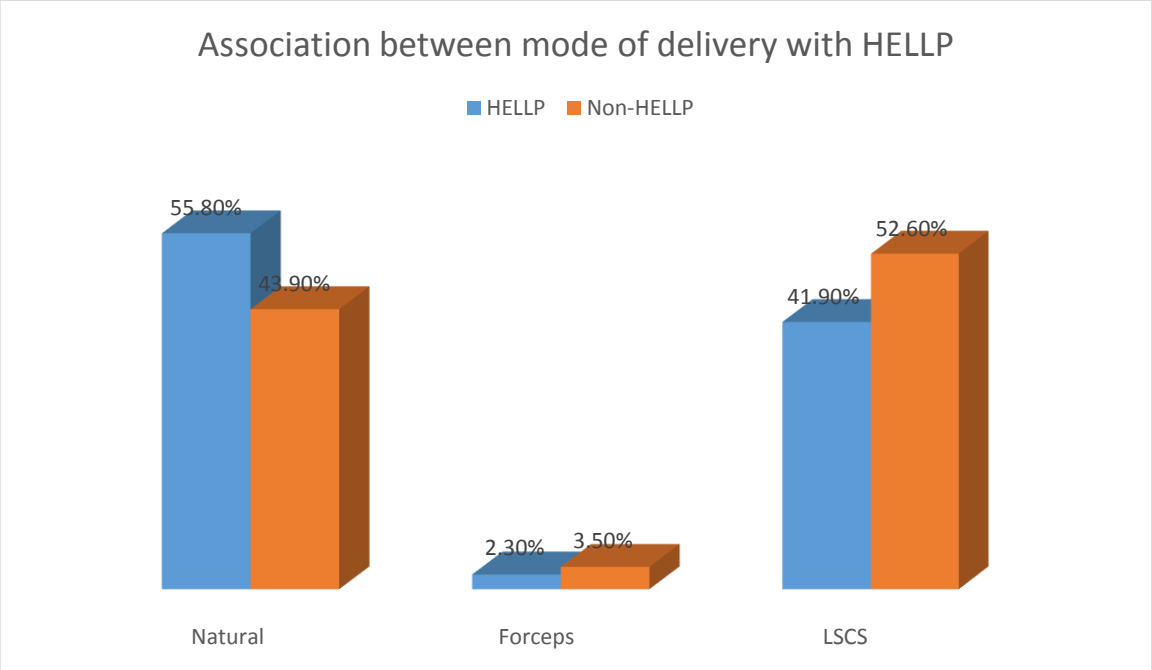
Term	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Pre term	36	83.7	37	64.9	73	73.0	4.399	1	P<0.05
Term	7	16.3	20	35.1	27	27.0			
Total	43	100.0	57	100.0	100	100.0			



83.7% of HELLP group had preterm delivery and 64.9% of Non-HELLP group had preterm delivery. The HELLP group were statistically associated with pre term delivery (P<0.05).

Table: 15. Association between mode of delivery with HELLP.

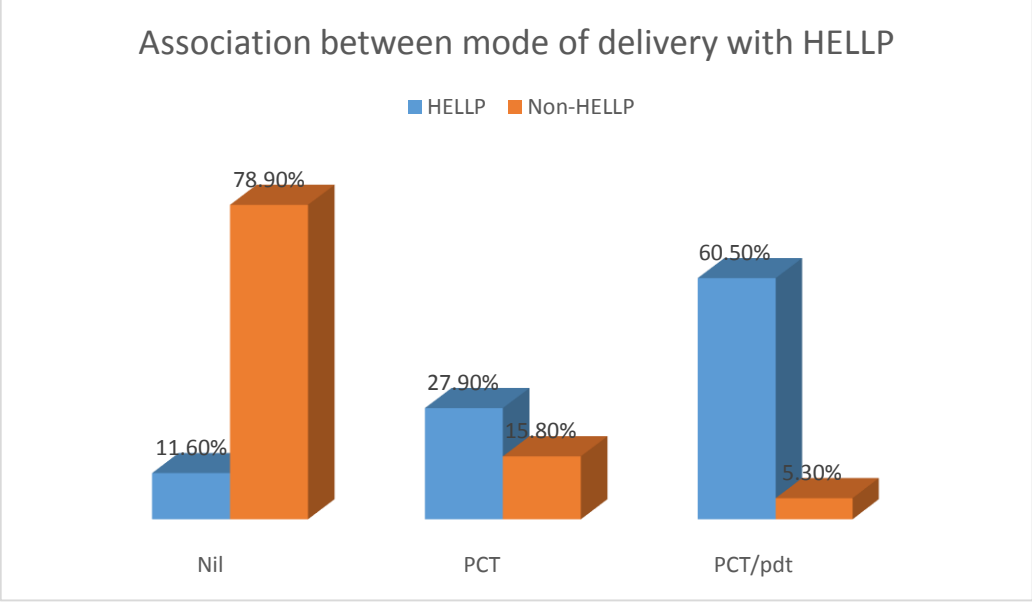
Mode of delivery	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Labour Natural	24	55.8	25	43.9	49	49.0	3.049	3	P>0.05
Forceps	1	2.3	2	3.5	3	3.0			
LSCS	18	41.9	30	52.6	48	48.2			
Total	43	100.0	57	100.0	100	100.0			



Among the HELLP group 55.8% delivered by LN, 2.3% by forceps, 41.9% by LSCS. Among the Non-HELLP group 43.9% delivered by LN, 3.5% by forceps, 52.6% by LSCS. There was no significant association between both groups. P>0.05.

Table: 16. Association between blood transfusion with HELLP.

Blood transfusion	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Nil	5	11.6	45	78.9	50	50.0	49.684	2	P<0.001
PCT	12	27.9	9	15.8	21	21.0			
PCT/pdt	26	60.5	3	5.3	29	29.0			
Total	43	100.0	57	100.0	100	100.0			



In the above table 84.4% of HELLP group required blood and blood product transfusion and only 21.1% of Non-HELLP group required blood transfusion. Hence there was need for blood transfusion in HELLP group which highly significant (P<0.001).

Table: 17. Type of Complications with HELLP and Non HELLP.

Complications	HELLP		Non-HELLP		Total	
	No	%	No	%	No	%
Nil	22	51.2	45	78.9	67	67.0
Abrubtio	9	20.9	6	10.5	15	15.0
ARF	7	16.2	2	3.5	9	9.0
Cerebral	1	2.3	1	1.8	2	2.0
CVT	1	2.3	1	1.8	2	2.0
DIC/Pulmonary Edema	1	2.3	0	0.0	1	1.0
Plueral/ effusion	1	2.3	1	1.8	2	2.0
PRES	0	0.0	1	1.8	1	1.0
SEPSIS	1	2.3	0	0.0	1	1.0
Total	43	100.0	57	100.0	100	100.0

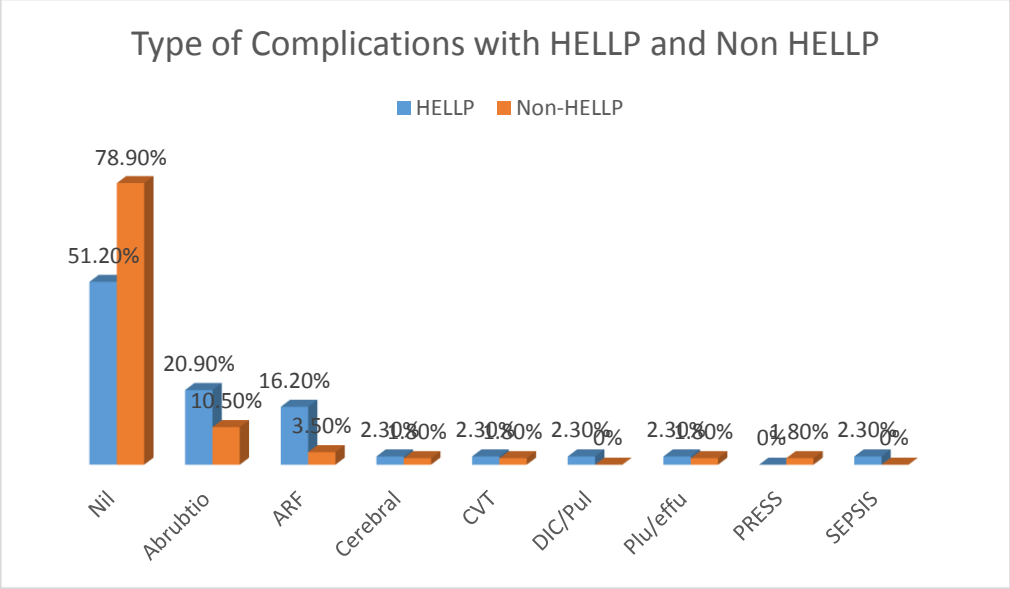
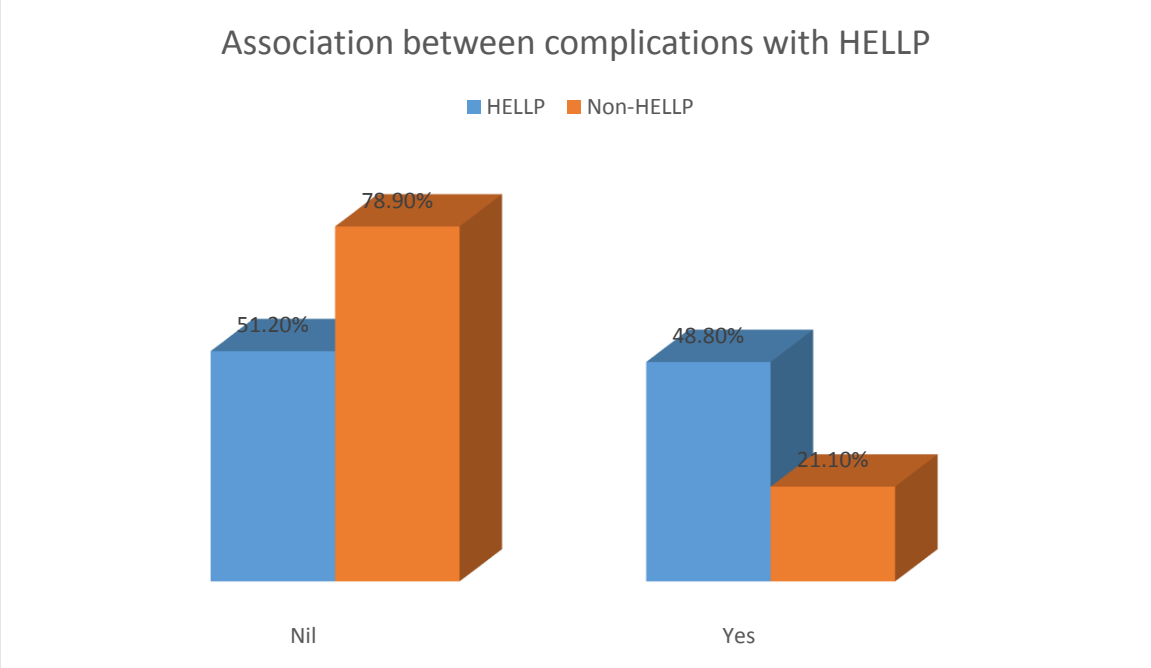


Table:18. Association between complications with HELLP.

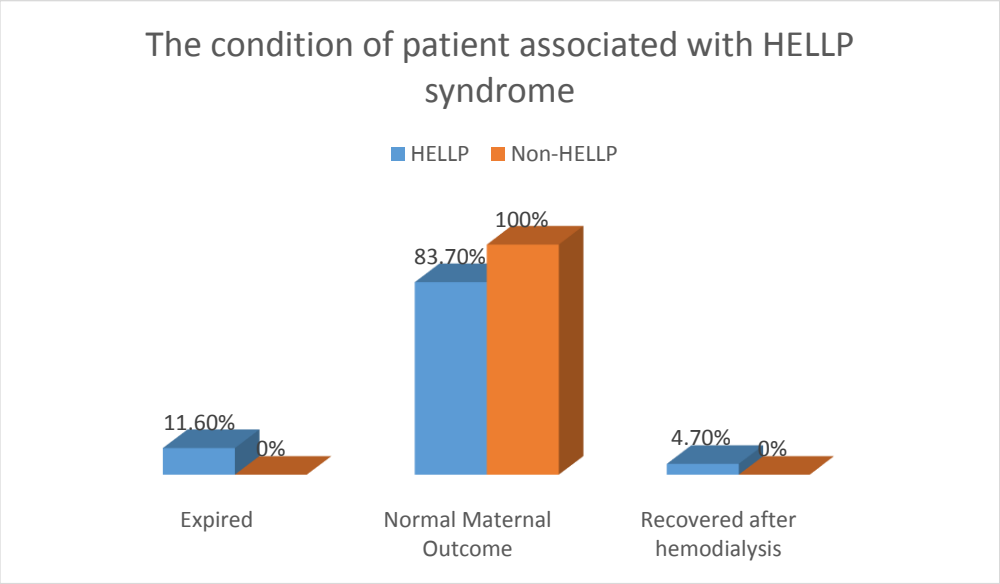
Complications	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Nil	22	51.2	45	78.9	67	67.0	8.558	1	P>0.01
Yes	21	48.8	12	21.1	33	33.0			
Total	43	100.0	57	100.0	100	100.0			



From above table 48.8% cases among the HELLP group developed complication and 21.1% cases among the Non-HELLP group developed complication. So the complication was strongly associated with HELLP syndrome group.

Table:19. The condition of patient associated with HELLP syndrome:

Condition	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
Expired	5	11.6	0	0.0	5	5.0	9.977	2	P<0.01
Normal Maternal Outcome	36	83.7	57	100.0	93	93.0			
Recovered after hemodialysis	2	4.7	0	0.0	2	2.0			
Total	43	100.0	57	100.0	100	100.0			



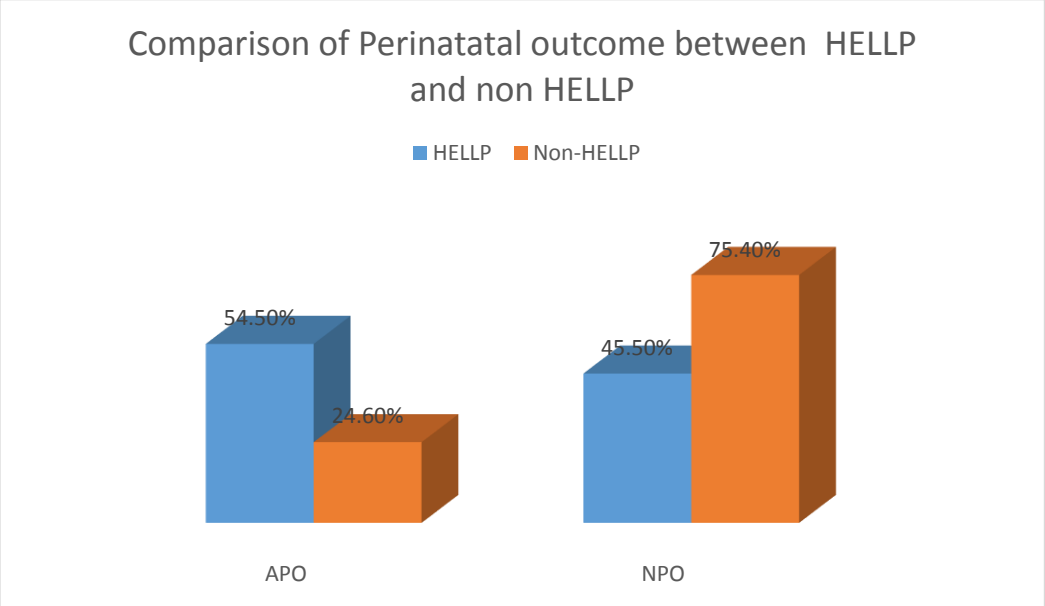
83.7% of case among HELLP group had normal maternal outcome and 100% of case among Non-HELLP group had normal maternal outcome. There was 11.6% mortality among the HELLP group hence mortality is strongly associated with HELLP syndrome.

Perinatal outcome:

The perinatal outcome was studied for Foetus. Among the HELLP group one patient had twins and all 42 mothers had singleton pregnancy. In HELLP group perinatal outcome was 44.

Table: 20.Comparison of Perinatal outcome between HELLP and non HELLP

Outcome	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
APO	24	54.5	14	24.6	38	37.6	9.513	1	P<0.001
NPO	20	45.5	43	75.4	63	62.4			
Total	44	100.0	57	100.0	101	100.0			



54.5% of babies among HELLP group had an abnormal perinatal outcome and 24.6% of babies among Non-HELLP group. Hence HELLP group is strongly associated with abnormal perinatal outcome. The association is statistically very highly significant (P<0.001).

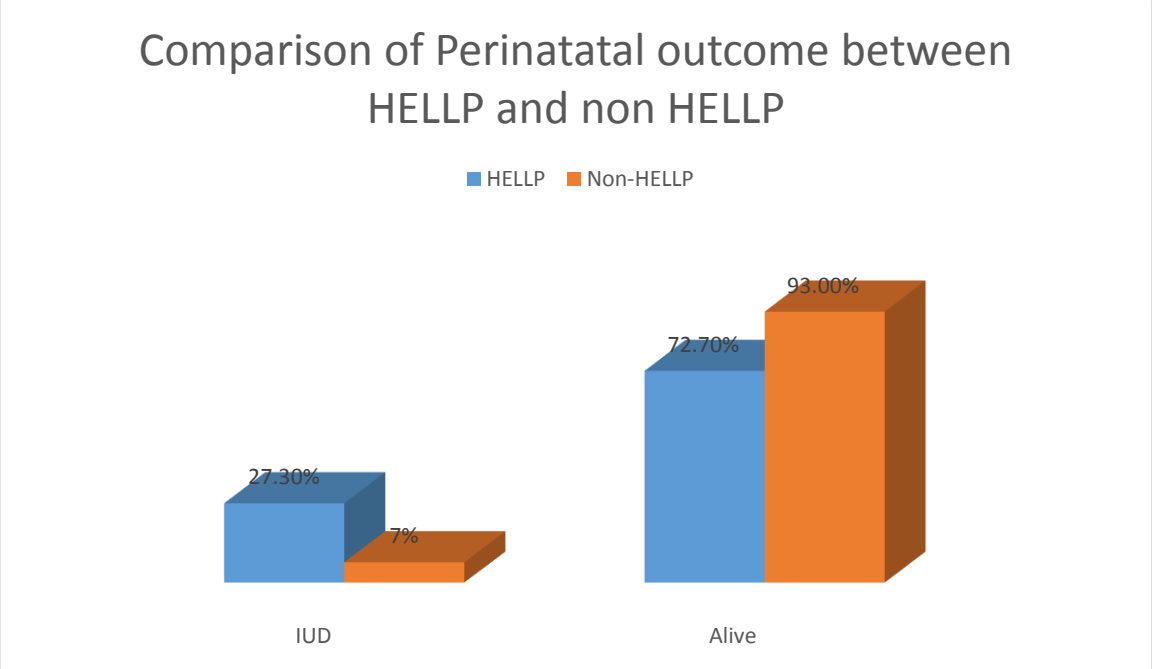


Table: 20.Comparison of Perinatal outcome between HELLP and non HELLP

Foetus condition	HELLP		Non-HELLP		Total		χ^2	df	Significance
	No	%	No	%	No	%			
IUD	12	27.3	4	7.0	16	15.8	7.642	1	P<0.01
Alive	32	72.7	53	93.0	85	84.2			
Total	44	100.0	57	100.0	101	100.0			

72.7% of babies among HELLP group were alive and 93% of babies among Non-HELLP group were alive. 27% were IUD among HELLP group and 7% among Non-HELLP group. Hence IUD is strongly associated with HELLP syndrome.

Table:21. Comparison of birth weight of babies between HELLP and non HELLP:

Variable	HELLP, n=44		Non HELLP, n=57		Difference b/w means	't'	df	Significance
	Mean	SD	Mean	SD				
birth weight	1.9	0.7	2.2	0.6	0.3	2.064	99	P<0.05

The mean birth weight of babies belonging to HELLP mothers was 1.9±0.7 Kg. The same of the non HELLP group mothers was 2.2±0.6 Kg. The different in the mean birth weights between the two groups was statistically significant (P<0.05).

Table:22 Treatment Regimen

	ANTI HYPERTENSIVES + $MGSO_4$ REGIMEN	ANTIHYPERTENSIVES ONLY
No. of cases	34	9
Percentage	79.0%	20.9%

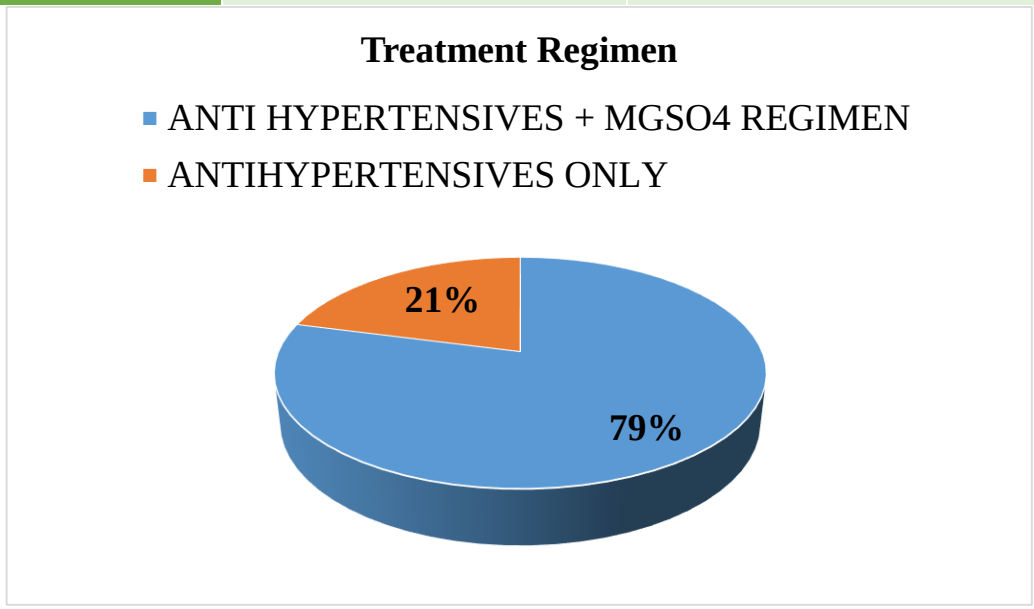
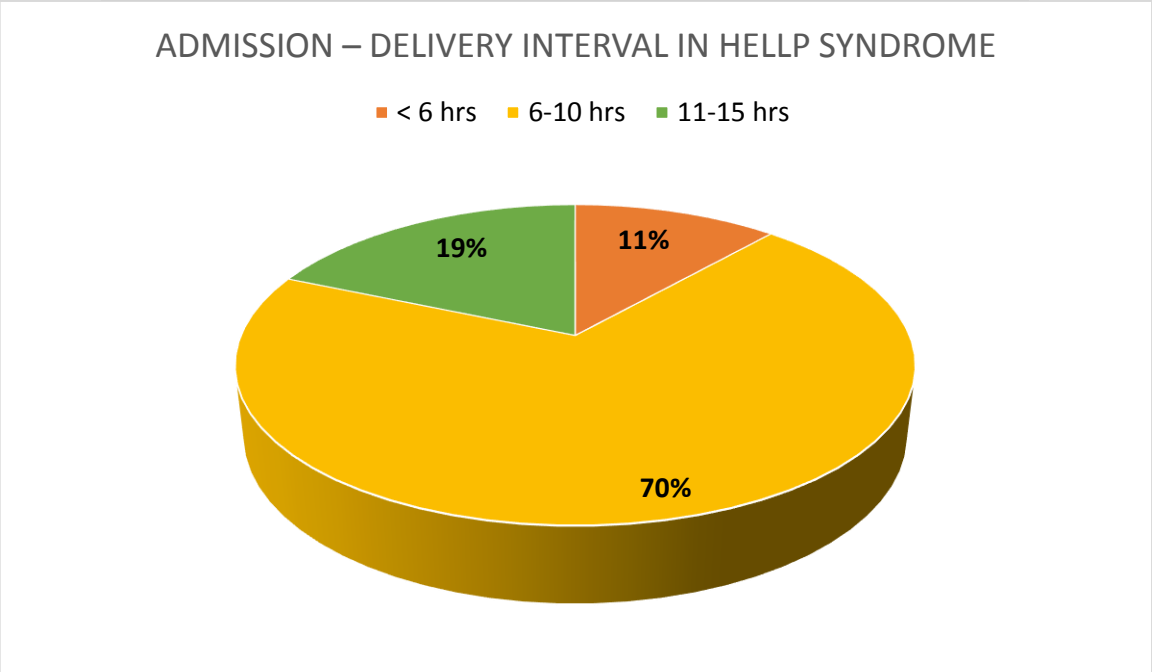


Table:23 ADMISSION – DELIVERY INTERVAL IN HELLP SYNDROME

< 6 hrs	6-10 hrs	11-15 hrs
5	30	8
11.6%	69.7%	18.6%



I ANALYSIS OF MATERNAL MORTALITY

a. AGE

20-24	25-29	30 and above
2	-	3
40%	-	60%

b. GRAVIDA

Primi	Multi
2	3
40%	60%

c. BLOOD PRESSURE

SEVERE PRE ECLAMPSIA	ECLAMPISA
4	1
80%	20%

d. GESTATIONAL AGE

28-30weeks	31-34 weeks
1	4
20%	80%

e. PROTEINURIA

++	+++	++++
1	3	1
20%	60%	20%

f. PLATELETS COUNT

CLASS I < 50,000	CLASS II 50,000 - 1,00,000	CLASS III 1,00,000 - 1,50,000
3	2	-
60%	40%	-

g. LDH

Case	LDH Level IU/L
1.	2242
2.	2774
3.	3000
4.	4000
5.	3686

All cases were between 2000-4000 IU/L

h. SGOT & SGPT

Cases	**SGOT**	**SGPT**
1.	114	96
2.	185	456
3.	176	576
4.	261	265
5.	256	254

There is high level of SGOT & SGPT in HELLP syndrome.

i. MODE OF DELIVERY

All patients delivered by LSCS.

j. ADMISSION-DELIVERY-DEATH INTERVAL

24 hrs	**48 hrs**
3	**2**
60%	**40%**

k. MATERNAL MORTALITY - CAUSES

ACUTE RENAL FAILURE WITH DIC	ABRUPTION WITH DIC	ABRUPTION WITH PULMONARY EDEMA
2	2	1
40%	40%	20%

According to Table a-k, there were 5 cases of death in HELLP syndrome giving rise to maternal mortality rate of 11.6%. 40% were primi between 20-24 years of age, and 60% were multi between 30-35 years of age mostly in severe preeclampsia at 30-34 weeks gestation. All cases had significant proteinuria, delivered within 15 hrs of admission. Most common causes of death were abruption and acute renal failure.

CONSOLIDATED ANALYSIS OF THE STUDY

STUDY		HELLP SYNDROME	NON- HELLP SYNDROME
Total No. of cases		43	57
Proteinuria		ALL	ALL
Fundus Changes		7	10
Low plateletcount		43	-
Evidence of haemolysis		43	-
Elevated liver enzymes		43	-
Mode of Delivery			
LN		24	25
Forceps		1	2
LSCS		18	30
Fetal outcome		12	
IUD			4
Alive		32	53
Preterm		36	37
Term		7	20
Maternal outcome			
No complication		22	45
Abruption		9	6
ARF		7	2
CVT		1	1
Cerebral edema		1	1
Pleural effusion		1	1
Sepsis		1	0
Pulmonary edema		1	0
HELLP SYNDROME	**Alive**	38	37
	Expired	5	0

CONSOLIDATED INFERENCE OF THE STUDY

Total no. of cases studied	:	100
Proteinuria	:	All (100%)
Fundus changes	:	17 (17%)
Low platelets count	:	43%
Elevated liver enzymes	:	43%
Evidence of haemolysis	:	43%

MODE OF DELIVERY

LN	:	49 (49%)
Forceps	:	3 (3%)
LSCS	:	48 (48%)

MATERNAL OUTCOME

Abruption	:	1 (15%)
Acute Renal failure	:	9 (9%)
CVT	:	2 (2%)
Cerebral edema	:	2 (2%)
Sepsis	:	1 (1%)
Pulmonary edema	:	1 (1%)
Pleural effusion	:	2 (2%)

PERINATAL OUTCOME

IUD	:	16 (16%)
Alive	:	85 (85%)
Preterm	:	73 (73%)
Term	:	27 (27%)

MATERNAL MORTALITY

IN HELLP SYNDROME : 5 (11.6%)

PERINATAL MORTALITY

IN HELLP SYNDROME : 24 (54.5%)

DISCUSSION

A study of 100 cases of HELLP syndrome and non-HELLP syndrome was undertaken during the year 2015 for a period of six months and the result were compared between the economic, demographic, physiological, biochemical, and obstetric character.

Incidence

James N.Martin	4-12%
B.E. Reubnoff	12-13.6%
Sibai 1986	9.7%
Sibai & Mohammed 1993	18.9%
My study	17.6%

The wide range of incidence can be attributed to the remarkable variability of the diagnostic criteria of the syndrome. In addition, these incidence rates cannot be considered for general population since they are reported from tertiary referral centres

Age

University of Tennessee, Memphis	24.4 years
English literature	24.9 years
My study	25.2 years

Gravida

B.E. Reubinoff (1990)	Multi
My study	Multi (55.8%)

High incidence of HELLP Syndrome in multi (55.8%)

Socioeconomic status

All cases belong to low socioeconomic status probably because our institution caters of mainly to economically deprived strata of society.

Gestational age

The average, gestational age at presentation is 33.8 weeks, 70% of HELLP Syndrome patients manifest before labour, while 30% manifest after delivery. According to Sibai 82% cases developed HELLP syndrome at <37 weeks. According to my study average gestational age at presentation is 33.2 weeks, HELLP syndrome manifest mostly during antepartum.

Clinical Symptoms & Signs

The frequent symptom is epigastric pain and /or right upper quadrant pain (90%) accompanied by nausea and vomiting. Headache may be present in 50% cases. In my study 40% cases had Headache associated with nausea and vomiting, rest of the patients (60%) with Oliguria, Nausea, Vomiting and convulsion.

Blood pressure

	Severe pre eclampsia	**Eclampsia**
Sibai et al	30%	10%
B.E. Reubinoff 1991	67%	-
My study	**90.7%**	**9.3%**

According to my study, 90% HELLP syndrome was associated with severe preeclampsia and 9.3% belonged to eclampsia.

Proteinuria

Urine analysis showed proteinuria of more than 2+ in 95.3% cases of HELLP Syndrome. It should be emphasized that 15% of HELLP Syndrome patients present with neither hypertension nor significant proteinuria (B.E. Reubinoff et al) In my study 4.7% patients had 1+ poteinuria, 30.2% had 2+ proteinuria, 48.8% had 3 + proteinuria and 16.3% had 4+ proteinuria.

According to Sibai, some women with HELLP Syndrome however hypertension and proteinuria may be absent or slight. Thus it is imperative that all health care providers become knowledgeable about clinical signs & symptoms that might herald the onset of HELLP Syndrome.

Platelets levels in HELLP Syndrome

	Class I	**Class II**	**Class III**
Martin 1990	1.09%	28%	71%
My study	**30.2%**	**55.8%**	**14%**

The definition of abnormal level varies among different studies. Severity of HELLP Syndrome is reflected in its laboratory parameters and not in the usual clinical parameters like blood pressure and proteinuria.

Haemolysis

Although microangiopathic hemolytic anemia underlies HELLP Syndrome, paradoxically most patients are not anemic when first admitted to the hospital. In my study all 43 cases had hemolytic blood picture with mean haemoglobin level of 7.8 gms ± 2.5g%.

Liver enzymes

According to B.E. Reubinoff et al., type of enzymes and definition of abnormal level also varies among different studies. There is direct correlation between the degree of thrombocytopenia and measures of liver dysfunction. An inverse correlation between platelets and LDH concentration was seen in both classes of HELLP Syndrome. Serum concentration of SGOT generally paralleled lactate dehydrogenase during the course of HELLP Syndrome. In my study there is inverse correlation between platelets and LDH in HELLP Syndrome.

Fundus Changes

In my study 7 cases of HELLP Syndrome had evidence of vasospasm and disc hyperemia at fundus examination (15.3%)

Treatment regimen

79% cases were started on $MgSO_4$ regimen in addition to antihypertensive, while 20.9% cases were in antihypertensive only.

Mode of delivery

According to Sibai (1999), caesarean delivery rate is high with HELLP Syndrome especially when pregnancy is less than 34 weeks of gestation (68%). In pregnancies less than 30 weeks of gestation caesarean section rate is 87%. According to my study, 55.8% cases of HELLP Syndrome delivered by Labour natural, 41.9% by LSCS 2.3% by Forceps.

Admission Delivery interval

According to Martin (1990) Length of time between hospital admission and delivery with a mean of 15.6 hours. According to my study admission delivery interval was less than 15 hours in all cases of HELLP Syndrome.

Maternal Outcome

Though coagulapathy has been mentioned as the most common complication of HELLP Syndrome, in my study all parameters like bleeding time (B.T), Clotting time (C.T), serum fibrinogen were normal. This may be due to non-sensitive parameters to detect DIC. More sensitive parameters like antithrombin IV, factor VIII and D-dimer may be needed to detect DIC.

	Abruption	Renal failure	Cerebral edema
Sibai 1999	20%	8%	1%
My study	**20.9%**	**16.2%**	**2.3%**

Maternal Mortality

According to sibai (1999) incidence of maternal mortality is as high as 24%. In my study, there were 5 cases of maternal death giving rise to maternal mortality rate of 11.6%, of these 1 case was due to Pulmonary edema with abruption 20%, 2 case was due to abruption and DIC 40% and 2 case was due to ARF with DIC 40%.

Analysis of maternal mortality

2 cases were in 20-25 years of age and 3 cases were in 30-35 years of age

Gravida - 2 cases were primigravida and 3 case were multigravida

Blood pressure-80% cases belong to severe preeclampsia, 20% cases belong to Eclampsia.

Gestational age - 4 cases were in 30-34 weeks and 1 case was in 28 weeks.

Proteinuria - All 5 patients had ≥ 2+proteinuria.

Platelet count - 40% cases belong to class-II and 60% cases belongs to class-I HELLP Syndrome.

Liver enzymes - SGOT, SGPT and LDH were significantly elevated in all 5 cases.

Mode of delivery – All delivered by LSCS.

Admission - delivery death interval - 1 day for 3 cases (60%) and 2 days for 2 case (40%) In spite of early delivery there was 5 deaths in my study, probably due to late referral.

Perinatal outcome

According to Sibai perinatal mortality is 30-40% primarily because of prematurity. There is a significant trend for advanced form of HELLP Syndrome (Class I & Class II) to appear at earlier gestational age. According to my study perinatal mortality is 54.5% primarily of prematurity.

Sibai 1999	30-40%
My study	54.5%

Birth weight of the baby

According to English literature, average weight of the new born is 1524-1898 gms and 30% were small for gestational age. According to my study the birth weight of live born babies of HELLP syndrome mothers significantly lesser than the non-HELLP syndrome babies. Mean birth weight of babies of HELLP mothers was 1.9 ± 0.7 kg.

Post-partum period

Lowest platelet count did not predict peak values of aspartate aminotransferase or lactate dehydrogenase. The degree of abnormality of platelet counts, AST and LDH did not perspectively and accurately predict time of recovery. According to text, at 5-7 days after delivery, platelet count were above 1,00,000/cumm in 85-96% of cases, AST were below 70 IU/L in 85-96% and LDH values were below 600 IU/L in 76-89%. In my study all cases for platelet count and liver enzymes reverted to normal levels by $4^{th} – 7^{th}$ post partum day.

1. The Hb level was significantly lesser among the HELLP syndrome mothers than their counter part non HELLP mothers.
2. The SB-T, D, and I were significantly more among the HELLP syndrome mothers than the non HELLP mothers.
3. Significant proteinuria is present in HELLP syndrome
4. The incidence of HELLP syndrome is significantly more among Multi gravida.

5. Gestational age of HELLP syndrome was significantly lesser than the non HELLP.
6. The SGOT, SGPT and LDH were significantly greater in HELLP group than the non HELLP group and Platelet count was significantly lesser in HELLP group than the non HELLP group.
7. HELLP syndrome more commonly occurs in preeclamptic patient
8. Mode of delivery was not significantly altered between both groups.
9. The blood transfusion was significantly essential to HELLP syndrome subjects.
10. The complications (48.8%) were significantly more among the HELLP group than the others.
11. The mortality incidence (11.6%) was significantly more among HELLP group.
12. The birth weight of live born babies of HELLP syndrome is 1.9 was significantly lesser than the other babies' birth weight.
13. 27.3% of IUD and 54.5% of abnormal prenatal outcome was strongly associated with HELLP syndrome.

SUMMARY

The HELLP syndrome and non-HELLP syndrome were compared and analysed for a period of six months from Feb2015 to July2015 and the summary is as follows.

Multigravida has a slightly higher incidence (55.8%) than primi.

Mean maternal age of HELLP Syndrome as per this study is 22.2 years.

Average gestational age that HELLP Syndrome presents is 33.2 weeks.

Most cases of HELLP Syndrome occurred during antepartum

Most cases of HELLP Syndrome belonged to severe preeclamptic group (90.7%)

95.3% cases of HELLP Syndrome had ≥ 2+ proteinuria.

30.2% cases of HELLP Syndrome belonged to class I, 55.8% cases of HELLP Syndrome belonged to class II and 14% cases belonged to class III HELLP Syndrome.

Mode of delivery - 58.1% cases of HELLP Syndrome delivered by labour natural, 41.9% by LSCS, 2.3% by forceps.

Admission delivery interval was <15 hours in all cases.

Significant maternal morbidity in HELLP Syndrome was due to abruption(20.9%) and acute renal failure (16.2%). Significant peritnatal morbidity and mortality in HELLP Syndrome was due to prematurity. Maternal mortality rate in HELLP Syndrome is 11.6% Perinatal Mortality rate in HELLP Syndrome is 54.5%. In all cases, the laboratory parameters returned to normal limits within 4^{th} – 7^{th} postpartum day.

CONCLUSION

The question of whether HELLP Syndrome exists as a distrint entity or not is a part of a spectrum of pregnancy complications, which have common liver dysfunction, haemolysis and thrombocytopenia has long been a source of speculation among obstetricians and physicians. However, the importance of this collection of signs and symptoms lies not in its name but rather in its associated high maternal and perinatal morbidity and mortality. Hence,

1. Patient with HELLP Syndrome warrants an emergency obstetric help.
2. HELLP Syndrome demands, careful and close evaluation of maternal and neonatal parameters and should be given equal attention in decision making.
3. Prompt delivery is mandatory regardless of gestational age.
4. Successful management requires recognition, a timely intervention and to render optimal patient treatment.

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PROFORMA

1. Name of the patient :
2. Age :
3. DOA :
4. D.O.D. :
5. IP No :
6. Socio Economic Group :
7. Gravida :
8. Para :
9. Abortion : 10. L.M.P. :
11. EDD :
12. Past H/o. :
13. F/H HT :

    Cardiac disease:

    D.M :
14. Present illness :

    a. Nausea / Vomiting :

    b. Oliguria :

    c. Blurring in Vision:

    d. Head Ache :

    e. Epi gastric pain :

    f. Convulsion :
15. G/E

a. Anaemia :

b. Oedema :

c. Pulse :

d. BP :

e. CVS :

f. RS:

16. O/E

a. Ascites :

b. Any overdistension :

c. Height of uterus :

d. Head engaged (or) not :

e. F.H.:

f. Amount of liquor :

g. Abnormal Presentation :

h. Complication Abruption :

17. **Investigations**

a. Daily weight :

b. Haemoglobin :

c. Albumin Urine :

d Fundus examination :

e. Blood Urea :

f. Sr.Uric Acid :

g. Sr.Creatinine :

h. Coagulation profile :

1 PT :

2 aPTT :

3 Fibrinogen :

i. Peripheral Smear :

1 anisocytosis :

2 Poikilocytosis :

3 Schistocytosis :

4 Burr Cells:

5 Micro Spherocyte

j. Serum Bilirubin :

Total Bilirubin :

Direct Bilirubin :

k. SGOT :

SGPT :

Sr. LDH :

l. Platelet count :

m. USG :

18. Management :

a Control of BP with Anti HT

b Prevention of seizure with Inj. $MgSo_4$:

c Haemotheraphy :

i PCT :

ii Platelet :

iii FFP :

iv Cryo :

d Induction / Augmentation

19. Outcome of delivery:

Mode of delivery :

a. Natural delivery : :

b. Forceps :

c. L.S.C.S. :

20. Perinatal putcome

a Alive / Still born / IUD

b Term / Preterm

c Birth Wt.

d Apgar

ABBREVATION

1. SGOT - Aspartate Transaminase
2. SGPT - Alanine Transaminase
3. H - Head ache
4. C - Convulsion
5. V - Vomiting
6. UO - Reduced Urinary Output
7. N - Normal
8. M -Multigravida
9. P - Primigravida
10. PE - Preeclampsia
11. E - Eclampsia
12. AP - Ante Partum Eclampsia
13. IP - Intra Partum Eclampsia
14. PP - Post Partum Eclampsia
15. B - Blurring of Vision
16. HU - Haematuria
17. Wgt - Weight
18. SYM - Symptom
19. GA - Gestational Age
20. BP - Blood Pressure
21. BU - Blood Urea
22. SC - Serum Creatinine

23. UA - Uric Acid
24. Urine ALB - Urine Albumin
25. HB - Haemoglobin
26. SrBil - T - Serum Bilirubin - Total
27. D - Direct
28. I - Indirect
29. LDH - Lactate Dehydrogenase
30. PLT - Platelet
31. PS - Peripheral Smear
32. MOD - Mode of Delivery
33. BT/Pdt - Blood Transfusion / Product
34. COMPLI - Complication
35. T/PT - Term / Preterm
36. APG at 1mt - Apgar at 1mt
37. MO - Maternal outcome
38. PO - Perinatal outcome
39. NPO - Normal Perinatal outcome
40. APO - Abnormal Perinatal outcome
41. CVT - Cortical vein Thrombosis
42. PUL Edema - Pulmonary Edema
43. Plu effusion - Plueral effusion
44. HD - Hemodialysis
45. DIC - Disseminated intra Vascular Coagulopathy

46. MODS - Multiple Organ Dysfunction Syndrome

47. NHBP - Normal Haemogram Blood Picture

48. OBSCODE - Obstetric Code

49. IUD - Intra uterine death

50. AFI - Amniotic fluid index.

51. EGA - Estimated gestational age

52. S_aO_2 - Oxygen saturation.

53. PLGF - Placental growth factor

54. HLA-C - Human Leukocyte Antigen – C

55. INF - Interferon

56. TGF - Transforming growth factor

57. VEGF - Vascular Endothelial Growth Factor

S.No.	Name	Age	IP No	SES	OBS CODE	FUN DUS	PE / E	GA	SYM	WGT Kg	BP mm Hg	BU mg %	SC mg %	UA mg %	URINE ALB	HB gm %	Sr Bil mg %			SGOT Iu/L	SGPT Iu/L	LDH Iu/L	PLT Lakhs/mm^3	HELLP CASES	PS	MOD	BT / Pdt	COMPLI	T/PT	WGT in Kg	APG 1mt	MO	PO
																	T	D	I													
1	Jebaseela	19	30980	IV	P	N	AP	37	C	60	160/110	32	0.9	5	++	11.4	0.4	0.2	0.2	21	23	216	302000	Non HELLP	NHBP	LSCS	-		T	2.16	7/10	WELL	NPO
2	Amudha	24	27405	V	M	N	PE	36	H	59	140/90	29	0.6	3.9	+	6.5	1.9	0.9	1	107	86	680	66000	HELLP	Burrcells	LN	PCT		T	2.75	8/10	WELL	NPO
3	Sayed Ali Fathima	26	26919	III	M	N	PE	34	H	65	170/120	16	0.7	2.1	+	12.1	0.8	0.4	0.4	26	13	175	230000	Non HELLP	NHBP	LSCS	-		PT	2.4	7/10	WELL	NPO
4	Mari	19	28183	V	P	N	PE	36	V	66	140/90	19	0.6	3.2	++	8.9	4.1	1.3	2.8	85	112	595	49000	HELLP	Schistocytes	LSCS	PCT/Pdt		PT	2.8	8/10	WELL	NPO
5	Jebaselvi	20	29256	IV	P	N	PE	37	H	61	180/130	16	0.6	4.5	+	12.7	0.4	0.2	0.2	26	36	195	185000	Non HELLP	NHBP	LSCS	-		T	2.25	7/10	WELL	NPO
6	Malathi	20	29128	V	P	G-I	PE	38	C/B	55	190/90	16	0.6	4.4	+++	10.6	0.7	0.4	0.3	55	65	701	88000	HELLP	fragmented RBCs	LSCS	PCT	Abruption / CVT	PT	2.6	6/10	WELL	APO
7	Pandichelvi	23	7648	V	M	N	PE	35	HU	56	180/130	23	1.2	7.1	+++	6.6	2.9	1.5	1.4	67	142	2500	34000	HELLP	Schistocytes	LN	PCT/Pdt	DIC/ Pul Edema	IUD	2.7	-IUD	WELL	APO
8	Sahith Mary	32	8626	V	P	N	AP	34	H/C	54	150/90	26	0.8	5.3	+++	11.1	0.8	0.4	0.4	37	23	356	212000	Non HELLP	NHBP	LSCS	-	CVT	PT	2.1	7/10	WELL	NPO
9	Kani	20	7596	IV	P	N	AP	35	C	58	130/90	17	1	4.2	+++	4.6	1.2	0.8	0.4	40	38	285	230000	Non HELLP	NHBP	LSCS	PCT	Plu effusion	PT	2.5	7/10	WELL	NPO
10	Sugapriya	27	6876	V	P	N	AP	30	C	52	170/110	16	0.9	7.1	++	12	1.0	0.5	0.5	80	75	182	92000	HELLP	NHBP	LN	PCT		PT	0.9	4/10	WELL	APO
11	Mahalakshmi	23	6721	III	M	N	PE	34	V	54	150/100	20	0.9	7.9	+++	5.4	1.6	0.6	1	112	126	3396	48000	HELLP	Schistocytes	LN	PCT/Pdt		PT	2.4	7/10	WELL	NPO
12	Angalaeswari	29	26947	V	P	N	PE	32	V	58	160/110	18	0.6	6	++	11	1.6	0.6	1	13	28	156	250000	Non HELLP	NHBP	LN	-		PT	1.6	5/10	WELL	APO
13	Muthulakshmi	25	26606	IV	P	N	PE	37	↓UO	52	156/110	33	2.5	7.9	+++	8	0.6	0.3	0.3	84	75	2115	86000	HELLP	Schistocytes	LN	PCT	ARF	T	3	8/10	WELL	NPO
14	Lakshmi	32	13488	IV	P	N	PE	28	H/V	55	180/120	16	7	7	++++	12.6	1.6	0.4	1.2	107	122	779	90000	HELLP	burrcells	LN	-	-	PT	1	4/10	WELL	APO
15	Tavampettral	34	13922	V	M	N	PE	36	-	57	100/110	18	1.1	5.2	++	10.4	0.6	0.3	0.3	23	18	564	186000	Non HELLP	NHBP	LSCS	-	-	PT	2.4	7/10	WELL	NPO
16	Mahalakshmi	23	8690	III	M	G-I	PE	36	↓UO	50	160/120	23	1.2	7.9	+++	4.8	1.6	0.6	1	186	106	3396	51000	HELLP	Schistocytes	LN	PCT/Pdt		PT	2.4	5/10	WELL	APO
17	Revathi	25	8430	V	P	N	PE	36	↓UO	59	150/110	20	0.8	6	++	11.6	0.8	0.4	0.4	26	20	452	201000	Non HELLP	NHBP	LSCS	-	-	PT	2.6	8/10	WELL	NPO
18	Rama	26	10297	IV	P	N	PE	34	-	54	160/120	15	9	9.9	+++	9.87	0.6	0.2	0.4	18	12	457	268000	Non HELLP	NHBP	LSCS	-	-	PT	2.1	7/10	WELL	NPO
19	Ulagammal	22	12954	V	P	G-I	PE	37	H/B	59	190/130	22	0.8		++++	7	1.0	0.5	0.5	102	95	2123	79800	HELLP	Schistocytes	LSCS	PCT/Pdt	Abruption	PT	2.9	8/10	WELL	NPO
20	Vijayalakshmi	20	18477	V	P	N	PE	35	C	60	200/120	23	0.9	9	++++	8	1.6	0.4	1.2	65	93	705	52000	HELLP	burrcells	LSCS	PCT/Pdt	Abruption	PT	2.4	6/10	WELL	NPO
21	Jothimalar	27	17367	I V	M	N	PE	36	↓UO	55	180/130	27	0.7	3.7	++	6.5	0.7	0.4	0.3	36	30	490	320000	Non HELLP	NHBP	LN	PCT	-	PT	2.25	6/10	WELL	NPO
22	Chellammal	20	16628	IV	P	N	PE	29	H	54	160/110	18	1.1	2.8	++	8.4	0.5	0.3	0.2	40	20	286	220000	Non HELLP	NHBP	LN	PCT	-	PT	1	3/10	WELL	APO
23	Umayaparvathi	22	16234	V	P	N	AP	38	C	56	170/140	16	7	2.6	++	10.3	0.6	0.3	0.3	36	37	175	272000	Non HELLP	NHBP	LSCS	-	PRESS	T	3	8/10	WELL	NPO
24	Iyyammal	20	21734	III	P	N	AP	38	C	60	180/120	21	9	7	++	1.4	0.5	0.2	0.3	40	32	318	250000	Non HELLP	NHBP	LSCS	-	Abruption / CVT	T	3.4	7/10	WELL	NPO
25	Angel	25	31625	IV	P	N	PE	30	H	54	180/110	38	1.2	8.6	+++	7.6	2.4	0.6	1.8	93	105	920	67000	HELLP	fragmentedRBCs	LN	PCT/Pdt	ARF	PT	1.3	-IUD	WELL/HD	APO
26	Noornisha	26	31259	V	P	N	PE	29	↓UO	55	150/110	62	2.9	9.9	++++	9	0.4	0.2	0.2	30	15	750	92000	HELLP	burrcells	LN	PCT	ARF	PT	1.2	-IUD	WELL/HD	APO
27	Uma maheswari	27	14065	IV	P	N	PE	36	-	59	170/130	16	1	3.6	++	11.8	0.4	0.2	0.2	29	16	551	350000	Non HELLP	NHBP	LSCS	-	-	PT	2	7/10	WELL	NPO
28	mariyammal	29	16947	V	P	N	PE	28	-	60	180/120	19	1.1	7.1	+++	6.7	0.5	0.2	0.3	120	102	2680	85000	HELLP	Schistocytes	LN	PCT/Pdt	Abruption / CVT	PT	1.2	-IUD	WELL	APO
29	Kadaleswari	21	19721	V	M	N	PE	34	↓UO	53	150/110	28	1.4		++	8.8	1.3	0.5	0.8	96	92	810	70000	HELLP	Schistocytes	LSCS	PCT/Pdt	Plu effusion	PT	18	6/10	WELL	NPO
30	Santhanamari	25	9860	IV	P	N	PE	28	H	36	190/110	28	0.8	7.2	+	13.3	0.8	0.4	0.4	170	112	175	41000	HELLP	fragmentedRBCs	LSCS	PCT/Pdt	-	PT	1.1	5/10	WELL	APO
31	mallika	24	9620	V	M	N	PE	32	H	58	140/110	37	1.2	8.3	++	12	0.5	0.3	0.2	68	46	652	120000	HELLP	NHBP	LN	-	-	PT	1.6	6/10	WELL	NPO
32	Vasanthi	32	9919	V	P	N	PE	36	-	57	150/100	18	0.7	12.6	++	12.6	1.0	0.6	0.4	38	22	160	285000	Non HELLP	NHBP	LSCS	-	-	PT	2.1	7/10	WELL	NPO
33	maari	29	8875	V	M	N	PE	30	H	58	170/100	18	0.9	6.8	+	10.3	1.0	0.5	0.5	57	72	450	156000	Non HELLP	NHBP	LN		-	PT	1.4	4/10	WELL	APO
34	Maheswari	23	9533	V	M	N	PE	36	-	56	150/110	17	1	6.2	+++	8.4	1.9	0.9	1	76	89	1675	45000	HELLP	fragmentedRBCs	LN	PCT/Pdt	Abruption	PT	2.2	6/10	WELL	NPO
35	kaleswari	25	10086	IV	P	N	AP	37	C	57	170/110	16	0.7	7.1	+++	9	1.7	0.7	1	61	70	640	249000	Non HELLP	NHBP	LSCS	-	-	T	2.6	7/10	WELL	NPO
36	angaleswari	24	10387	III	M	N	PE	34	HU	58	140/100	17	0.6	6.8	+++	6.4	2.5	0.9	1.6	92	88	4253	32000	HELLP	Schistocytes	FORCEPS	PCT/Pdt	Abruption / pul edema	T	2.8	8/10	WELL	NPO
37	marithai	23	22184	IV	P	N	PE	34	-	56	170/100	19	0.6	8.8	++	8	2.9	0.2	2.7	96	115	1617	42000	HELLP	fragmentedRBCs	LN	PCT/Pdt	Abruption / sepsis	PT	1.6	-IUD	WELL	APO
38	maliyammal	27	22780	V	M	N	PE	28	H	53	170/100	2	0.8	3.8	+++	12.6	1.6	0.8	0.8	40	14	897	103000	HELLP	burrcells	LN	-	-	PT	1	4/10	WELL	APO
39	baby	2	10095	IV	P	N	PE	37	-	49	150/100	18	0.7	4	++	8	0.6	0.3	0.3	31	27	872	187000	Non HELLP	burrcells	FORCEPS	PCT/Pdt	-	T	2.8	7/10	WELL	NPO
40	esakkiyammal	21	9994	IV	P	N	PE	36	-	53	170/110	16	0.8	7.2	++	11.8	0.6	0.3	0.3	29	11	584	264000	Non HELLP	NHBP	LN	-	-	PT	2.6	8/10	WELL	NPO
41	megala	25	27790	IV	P	N	PE	35	-	51	150/110	18	0.7	4.6	++	12.5	0.4	0.2	0.2	27	24	176	211000	Non HELLP	NHBP	LN	-	-	PT	2	8/10	WELL	NPO
42	aruna saro	31	17154	III	M	N	PE	32	H	70	190/130	40	3.1	9.2	+++	7	6.0	3	3	114	96	2242	29000	HELLP	Schistocytes	LSCS	PCT/Pdt	ARF / DIC	PT	1.8	7/10	expired	APO
43	aladi	30	20500	IV	P	N	PE	38	-	62	140/100	16	0.6	6	++	14.4	1.7	0.7	1	30	70	620	99000	HELLP	fragmentedRBCs	LSCS	PCT/Pdt		T	3	8/10	WELL	NPO
44	rajam	33	25325	IV	M	N	PE	28	V/H	58	180/120	33	1.7	8.8	++	8.7	5.2	2	3.2	185	456	2774	29080	HELLP	Schistocytes	LSCS	PCT/Pdt	ARF / MODS	PT	1	-IUD	expired	APO
45	bramaraj sanjana	35	11765	IV	M	G-II	PE	30	B/H	76	220/140	56	3.2	10.3	++++	6.2	7.4	2.4	5	176	576	3000	89000	HELLP	Schistocytes	LSCS	PCT/Pdt	ARF / Pul edema	PT	1.2	-IUD	expired	APO
46	pannerselvi	25	9671	IV	P	N	PE	38	H	56	170/110	28	0.7	8.2	+++	9	1.0	0.7	0.3	30	28	125	125000	Non HELLP	NHBP	LSCS	PCT		T	3	8/10	WELL	NPO
47	mumdaj	26	12430	V	P	N	PE	32	H	60	160/120	20	0.6	6.2	+++	6	0.8	0.6	0.2	116	118	195	105000	HELLP	NHBP	LN	PCT		PT	1.7	7/10	WELL	NPO
48	kanniyammal	19	11915	IV	P	N	PE	28	V	50	160/120	26	0.7	7.8	+++	9.2	0.9	0.6	0.3	28	28	278	110000	Non HELLP	NHBP	LN	-		PT	1.1	-IUD	WELL	APO
49	pooma	23	18108	V	P	G-I	PE	34	H/B	66	170/120	31	1.9	9	+++	4	2.3	1.5	0.8	261	265	4000	40000	HELLP	Schistocytes	LSCS	PCT/Pdt	ARF	PT	2	2/10	expired	APO
50	muthumari	26	21254	IV	P	N	PE	38	-	66	170/120	28	0.8	8.4	+++	9	0.9	0.7	0.2	39	36	388	270000	Non HELLP	NHBP	LSCS	-		T	3.2	7/10	WELL	NPO
51	valli	28	22294	IV	P	N	PE	34	-	63	170/120	30	0.6	7.3	+++	6.5	0.9	0.7	0.2	96	94	215	215000	Non HELLP	NHBP	LN	PCT		PT	1.7	8/10	WELL	NPO

52	zareena	23	25438	IV	P	G-I	PE	36	H/B	55	180/120	68	1.4	7.5	++++	7.6	0.8	0.5	0.3	24	26	205	205000	Non HELLP	NHBP	LSCS	PCT	ARF	PT	1.6	3/10	WELL	APO
53	kavitha	17	26077	V	P	N	PE	38	H/V	60	170/120	30	0.7	7.6	+++	9	0.9	0.6	0.3	82	86	195	180000	Non HELLP	NHBP	LSCS	PCT/Pdt	Abruption	T	1.9	6/10	WELL	NPO
54	arumugam kani	27	2986	IV	P	G-I	PE	28	-	60	180/120	24	1	7.3	++++	8.6	0.8	0.6	0.2	26	28	68	130000	Non HELLP	NHBP	LN		Abruption	PT	1.1	-IUD	WELL	APO
55	brammachi	21	41158	IV	P	G-I	PE	37	-	49	170/110	24	0.6	8	+++	6.2	0.9	0.6	0.3	30	32	184	195000	Non HELLP	NHBP	LSCS			T	3.2	6/10	WELL	NPO
56	anna selvi	18	23448	IV	P	G-II	PE	36	-	51	150/120	23	0.7	6.8	+++	9	0.9	0.6	0.3	152	146	668	115000	Non HELLP	burrcells	LN			PT	2.1	8/10	WELL	NPO
57	lathamary	25	51389	V	P	N	PE	36	-	52	160/110	28	0.6	8.2	+++	6	0.8	0.5	0.3	22	24	118	205000	Non HELLP	NHBP	LN			PT	2.2	8/10	WELL	NPO
58	niraimathi	31	9824	IV	P	N	PE	36	-	66	170/110	38	0.6	7.3	+++	9.2	0.9	0.6	0.3	36	34	194	115000	Non HELLP	NHBP	LSCS			PT	1.9	8/10	WELL	NPO
59	thavamani	25	7038	V	P	N	PE	37	-	53	160/110	28	0.6	7.8	+++	10.1	0.9	0.6	0.3	20	28	107	135000	Non HELLP	NHBP	LN			T	2.4	8/10	WELL	NPO
60	grace	23	23894	IV	P	N	PE	36	-	49	170/120	28	0.6	8	+++	11.6	0.8	0.5	0.3	26	28	148	142000	Non HELLP	NHBP	LN			PT	1.8	8/10	WELL	NPO
61	kavipriya	21	29854	IV	P	G-II	PE	28	-	58	170/140	22	0.7	7.1	++++	6.2	0.9	0.6	0.3	182	186	695	78000	HELLP	burrcells	LN	PCT/Pdt		PT	0.8	-IUD	WELL	APO
62	mythili	28	34187	IV	P	N	PE	37	-	61	180/130	32	0.8	7.9	+++	8.6	0.8	0.5	0.3	91	93	212	165000	Non HELLP	NHBP	LSCS			T	2.8	8/10	WELL	NPO
63	leemarose	23	44185	IV	M	N	PE	36	-	59	170/120	33	1.4	9	+++	7.2	2.0	0.7	1.3	253	172	1240	40000	HELLP	Schistocytes	LSCS	PCT	sepsis	T	2.1	7/10	WELL	NPO
64	durgadevi	25	37288	V	M	N	PE	36		58	140/110	26	0.8	3.2	+++	5	1.1	0.5	0.6	230	115	1184	19000	HELLP	Schistocytes	LSCS	PCT/Pdt		PT	2.5	7/10	WELL	NPO
65	malathi	32	38305	IV	M	N	PE	37	H	57	160/120	38	1.5	9	++	6	1.2	0.5	0.7	124	112	2292	47000	HELLP	Schistocytes	LSCS	PCT/Pdt	CVT	PT	3.2	7/10	WELL	NPO
66	ushadevi	25	31873	IV	M	G-I	PE	30		60	160/120	22	0.6	7.1	+++	10	1.0	0.7	0.3	22	26	98	115000	Non HELLP	NHBP	LN			PT	1.2	7/10	WELL	NPO
67	banu	19	28562	IV	M	N	PE	37	H	62	170/110	28	0.6	8.2	+++	9.2	0.9	0.6	0.3	94	90	611	105000	HELLP	burrcells	LN			T	1.8	7/10	WELL	NPO
68	muneeswari	31	35010	V	M	GI	PE	38	↓UO	54	160/120	30	0.7	7.9	+++	11.1	0.8	0.5	0.3	28	94	118	195000	Non HELLP	NHBP	LSCS		Abruption	T	3.1	2/10	WELL	APO
69	devi	22	40035	V	M	N	PE	36	-	50	170/110	20	0.7	7	+++	11.2	0.9	0.6	0.3	28	26	138	125000	Non HELLP	NHBP	LN			PT	1.8	8/10	WELL	NPO
70	muppidathi	29	41095	IV	M	N	PE	39	H/V	61	160/120	26	0.6	6.3	++	11.4	0.8	0.5	0.3	28	26	132	150000	Non HELLP	NHBP	LN			T	2	2/10	WELL	APO
71	gomathi	20	52924	IV	M	N	PE	34	V	47	170/110	30	0.7	6.8	+++	10.6	0.9	0.6	0.3	30	28	184	110000	Non HELLP	NHBP	LN			PT	1.75	8/10	WELL	NPO
72	ambika	28	46003	IV	M	N	PE	32	H	52	150/110	23	0.6	7.6	+++	6	2.8	1.6	1.2	124	128	1652	51000	HELLP	Schistocytes	LN	PCT		PT	1.4	2/10	WELL	APO
73	helen	31	52924	V	M	N	PE	38	H	59	160/120	24	0.7	7.3	++	7	0.9	0.5	0.4	81	84	632	55000	HELLP	burrcells	LSCS,TWI	PCT		T	1.8,1.9	7/10	WELL	NPO
74	vijaya	23	57882	V	M	G-II	PE	28	H/V	55	150/120	20	0.7	7.3	+++	11.4	0.8	0.6	0.2	30	28	154	230000	Non HELLP	NHBP	LN	-		PT	1	-IUD	WELL	APO
75	kala	23	59023	V	M	N	PE	34	-	49	120/100	26	0.6	7.2	+++	5.2	0.9	0.6	0.3	84	86	620	70000	HELLP	burrcells	LN	PCT		PT	2.2	-IUD	WELL	APO
76	kaliammal	31	61785	IV	M	N	PE	34	-	54	120/110	26	0.6	6.8	+++	8	0.9	0.6	0.3	34	32	124	170000	Non HELLP	NHBP	LN	PCT		PT	1.7	8/10	WELL	NPO
77	eswari	27	62951	IV	M	N	PE	36	H	54	160/110	19	0.7	6.9	++	7.4	1.8	0.5	1.3	86	84	731	65000	HELLP	burrcells	LN	PCT/Pdt		PT	1.8	2/10	WELL	APO
78	krishnammal	25	6796	IV	M	G-I	PE	38	-	58	170/120	28	0.6	6.8	++	8.2	1.0	0.7	0.3	20	28	176	195000	Non HELLP	NHBP	FORCEPS	PCT/Pdt		T	3.3	8/10	WELL	NPO
79	chitra	29	5836	V	M	N	PE	32	H/V	62	160/120	18	0.7	6.5	+++	4.8	0.9	0.6	0.3	102	110	1027	81000	HELLP	fragmentedRBCs	LN	PCT/Pdt		PT	1.2	3/10	WELL	APO
80	ananthi	18	18108	IV	P	N	AP	28	H/C	56	160/120	18	0.7	6.4	++++	6.6	0.9	0.7	0.2	75	78	1652	85000	HELLP	fragmentedRBCs	LN	PCT		PT	0.9	-IUD	WELL	APO
81	ammulakshmi	20	37985	IV	P	G-I	AP	34	H/V	48	140/110	24	0.9	8.2	+++	6	3.9	1.6	2.3	256	254	3686	75000	HELLP	burrcells	LSCS	PCT/Pdt	Abruption / DIC	PT	1.8	-IUD	expired	APO
82	ramalakshmi	24	7488	IV	P	N	AP	28	H/V	66	150/100	22	0.6	7.1	+++	10.4	0.8	0.5	0.3	24	24	162	165000	Non HELLP	NHBP	LSCS	-		PT	0.85	1/10	WELL	APO
83	vennila	31	4829	V	M	N	AP	39	C	60	160/110	18	0.7	7.2	++	11.6	1.0	0.7	0.3	80	80	256	185000	Non HELLP	NHBP	LSCS	-		T	2.7	8/10	WELL	NPO
84	sakthi	22	2243	V	M	N	PE	38	C	49	150/110	22	0.8	7.6	++	8.6	0.9	0.6	0.3	82	86	128	115000	HELLP	NHBP	LN	PCT		T	2.6	8/10	WELL	NPO
85	sumathi	22	28492	V	M	N	AP	38	C	52	160/110	20	0.6	6.1	+++	71.1	0.9	0.6	0.3	74	48	192	225000	Non HELLP	NHBP	LSCS	-		T	2.8	7/10	WELL	NPO
86	danalakshmi	23	31876	IV	P	N	AP	34	C	56	150/110	22	0.7	6.4	+++	10.6	0.9	0.7	0.2	18	19	112	115000	Non HELLP	NHBP	LSCS	-		PT	2	7/10	WELL	NPO
87	vadivu	24	49320	V	M	N	AP	34	C	62	140/100	20	0.6	7.1	+++	9.4	0.8	0.5	0.3	19	17	102	165000	Non HELLP	NHBP	LSCS	-		PT	1.9	8/10	WELL	NPO
88	rosey	20	56301	V	P	N	AP	36	C	46	140/100	20	0.6	6.8	+++	9.2	0.8	0.6	0.2	92	96	126	115000	HELLP	NHBP	LSCS	-		PT	2.8	8/10	WELL	NPO
89	mageswari	27	67925	V	P	G-I	AP	30	C	50	150/110	22	0.7	7.1	++	9	0.9	0.6	0.3	20	21	140	125000	Non HELLP	NHBP	LN	-		PT	.1.4	2/10	WELL	APO
90	sundarammal	31	58630	IV	M	N	AP	28	C	59	160/100	18	0.6	7	++	5.1	3.9	1.6	2.3	98	112	1028	52000	HELLP	fragmentedRBCs	LSCS	PCT/Pdt	cerebraledema	PT	1.1	1/10	WELL	APO
91	sowmiya	32	59023	V	M	N	AP	34	C	63	140/110	22	0.7	7	+++	7.4	0.9	0.6	0.3	19	22	96	140000	Non HELLP	NHBP	LSCS	PCT	cerebraledema	PT	2.1	7/10	WELL	NPO
92	malar	23	2136	V	P	N	AP	38	C	58	150/100	18	0.8	6.5	++	11.5	0.8	0.5	0.3	24	26	102	220000	Non HELLP	NHBP	LSCS	-		T	3	8/10	WELL	NPO
93	piriyanka	18	6023	IV	P	N	PE	36	H/V	47	150/110	44	1.6	8	++	11.5	0.9	0.6	0.3	34	30	146	105000	Non HELLP	NHBP	LN	-	ARF	PT	2.1	-IUD	WELL	APO
94	rajeswari	22	3242	V	M	N	PE	32	H	54	170/100	22	0.7	7	+++	11.2	0.8	0.5	0.3	26	22	154	115000	Non HELLP	NHBP	LN	-		PT	1.4	7/10	WELL	NPO
95	buela	28	3949	V	M	N	PE	37	V	65	150/100	24	0.6	3.7	++	6.4	0.8	0.5	0.3	20	24	126	126000	Non HELLP	NHBP	LSCS	PCT	Abruption	PT	2.9	8/10	WELL	NPO
96	sangari	23	35186	IV	P	N	PE	28	V	57	160/100	20	0.6	7.3	++	10.6	0.9	0.6	0.3	32	24	138	195000	Non HELLP	NHBP	LN	-		PT	1.1	1/10	WELL	APO
97	patturani	32	32018	IV	M	N	PE	30	↓UO /H	61	160/110	24	0.6	7.6	++	7.1	0.9	0.7	0.2	24	24	725	95000	HELLP	NHBP	LN	PCT/Pdt	Abruption	PT	0.9	IUD	WELL	APO
98	rathi	28	29870	V	P	N	AP	32	H/C	61	170/90	28	0.6	9	+++	11.4	0.8	0.5	0.3	28	30	185	310000	Non HELLP	NHBP	LN	-		PT	1	3/10	WELL	APO
99	muthu kani	30	41098	V	M	G-I	PE	37		54	160/120	26	0.6	9.2	+	12	0.9	0.6	0.3	28	28	176	250000	Non HELLP	NHBP	LSCS	-		T	2.5	8/10	WELL	NPO
100	sorna	24	5229	V	P	N	PE	34	H	60	160/110	32	0.7	8	++	10.8	0.9	0.6	0.3	24	22	202	210000	Non HELLP	NHBP	LN	-	Abruption	PT	1.7	8/10	WELL	NPO